The Journal
OF THE KANSAS MEDICAL SOCIETY

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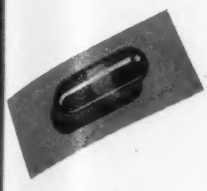

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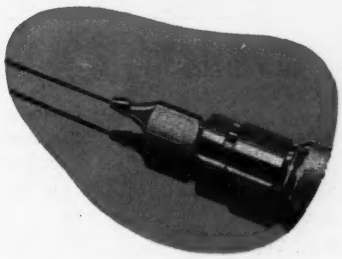

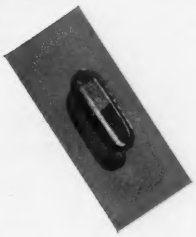

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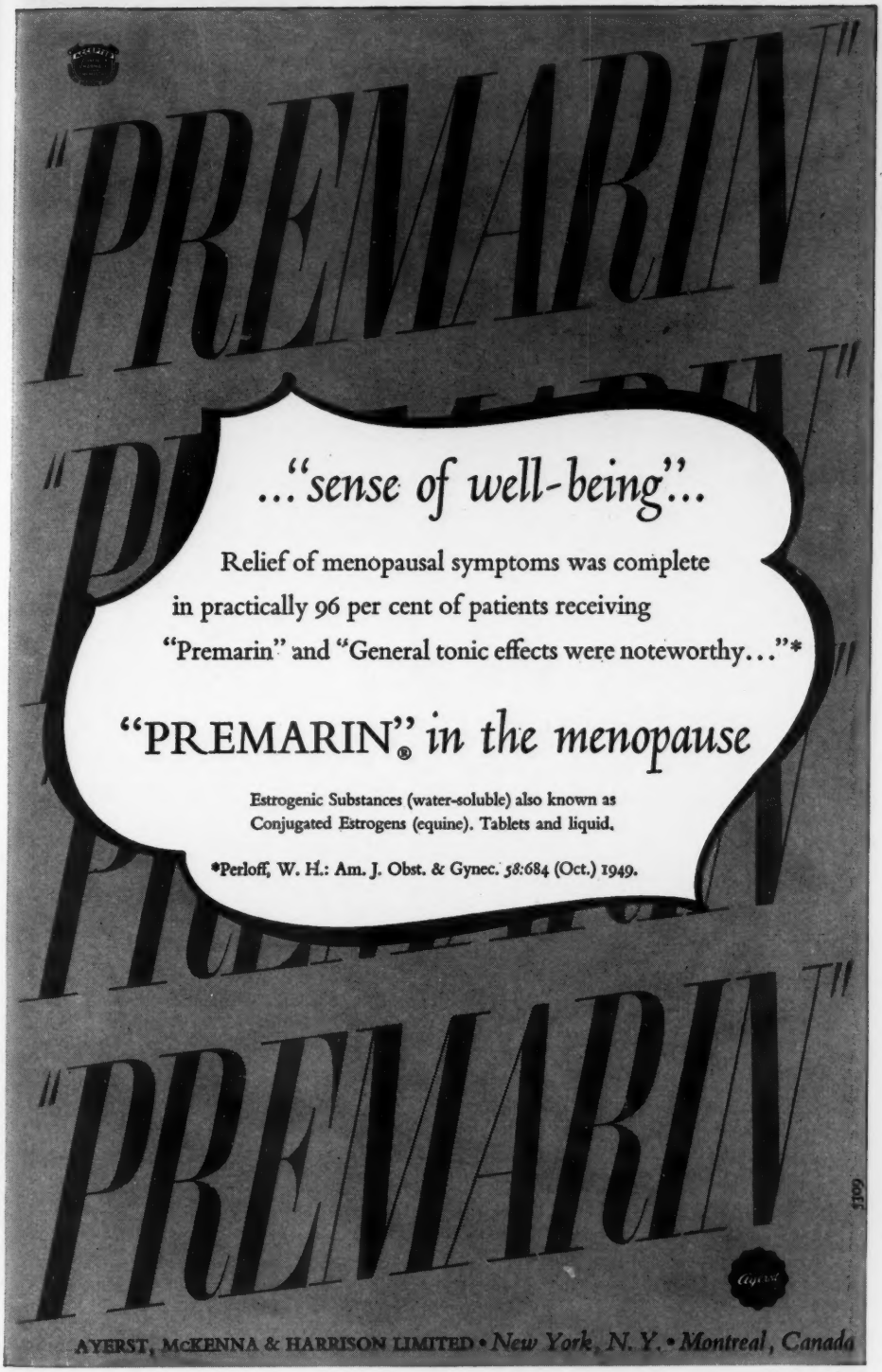
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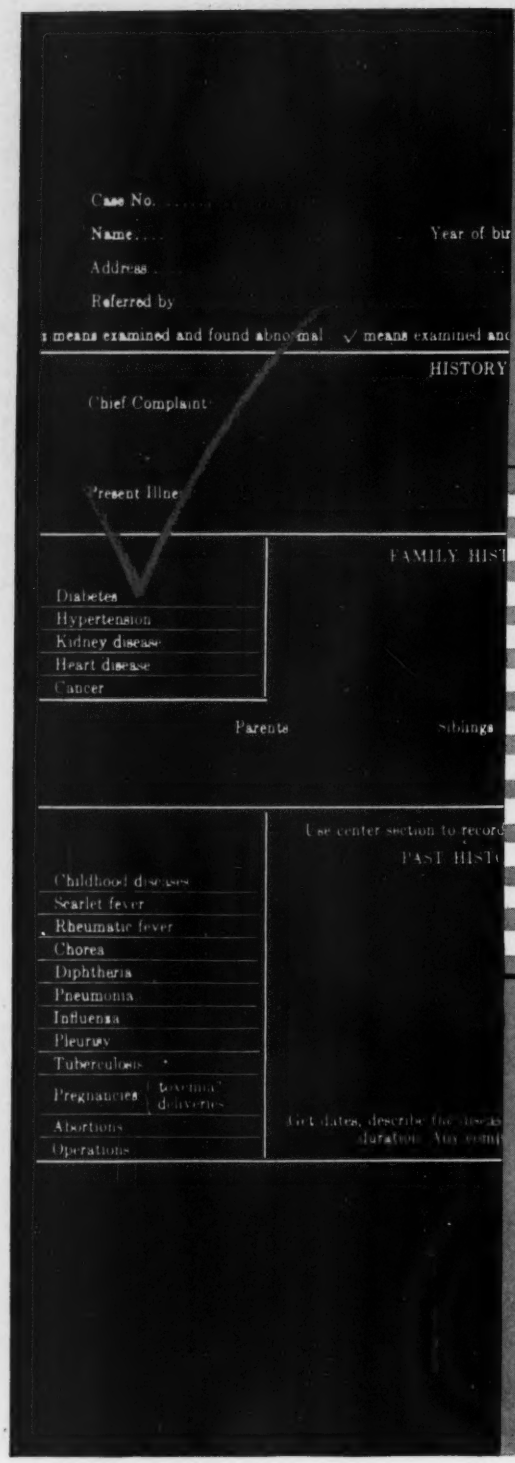

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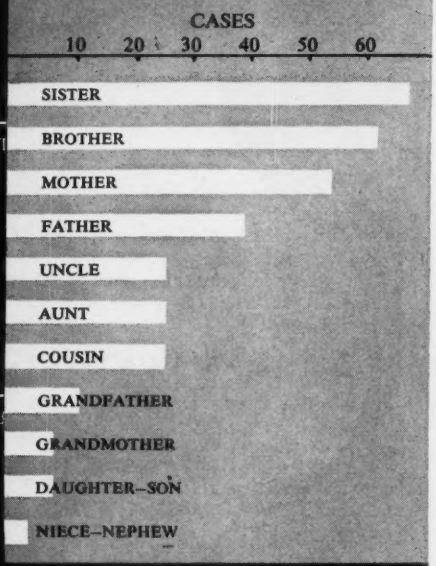

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2. Allen, F. M.: Diabetes Mellitus, in Piersol, G. M., and Bortz, E. L.: Cyclopedia of Medicine, Surgery, Specialties, Philadelphia, F. A. Davis Company, 1951, vol. 4, p. 505.

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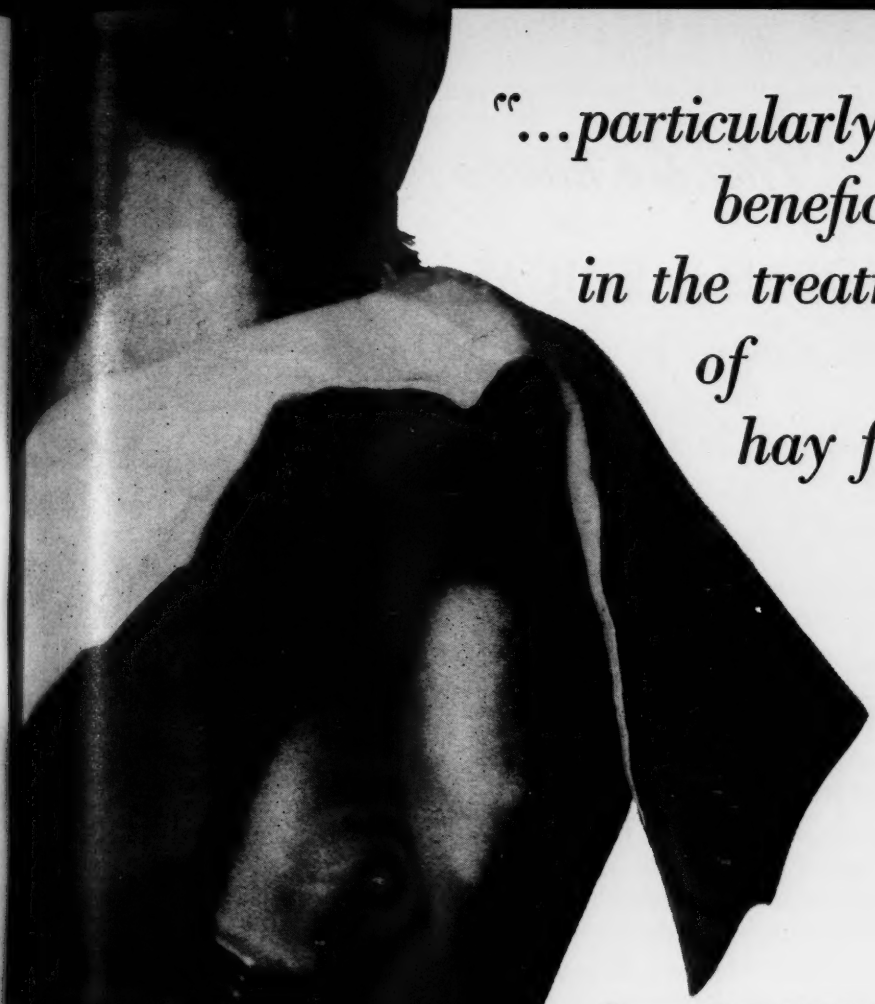

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1. Silbert, N. E.: New England J. Med. 242:931, 1950.
2. Eisenstadt, W. S.: Journal Lancet 70:26, 1950.

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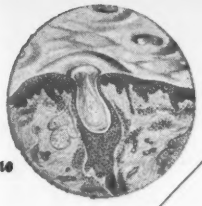

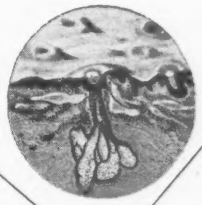

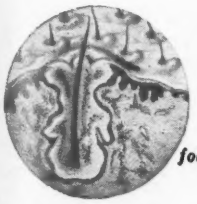

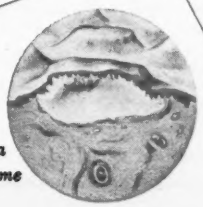

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1. *Bednar, G. A.: South. M. J. 46:298 (March) 1953.*
2. *Wright, C. S. et al.: A. M. A. Arch. Dermat. & Syph. 67:125 (Feb.) 1953.*
3. *Robinson, H. M. et al.: South. M. J. (in press).*
4. *Andrews, G. C. et al.: J. A. M. A. 146:1107 (July 21) 1951.*

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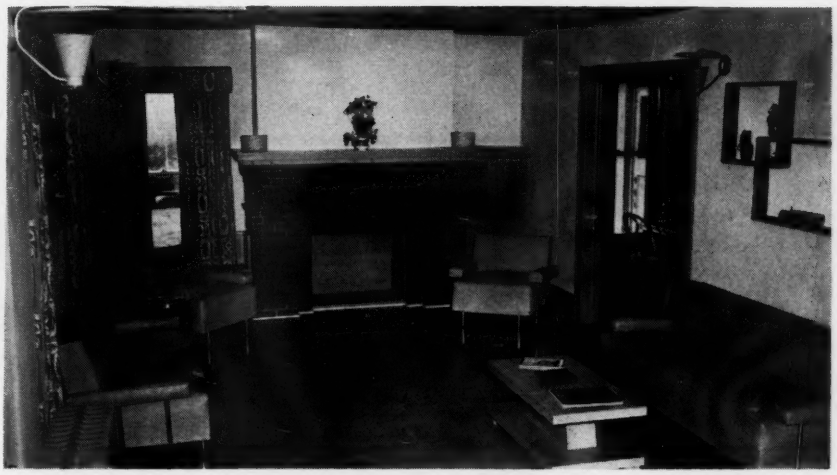

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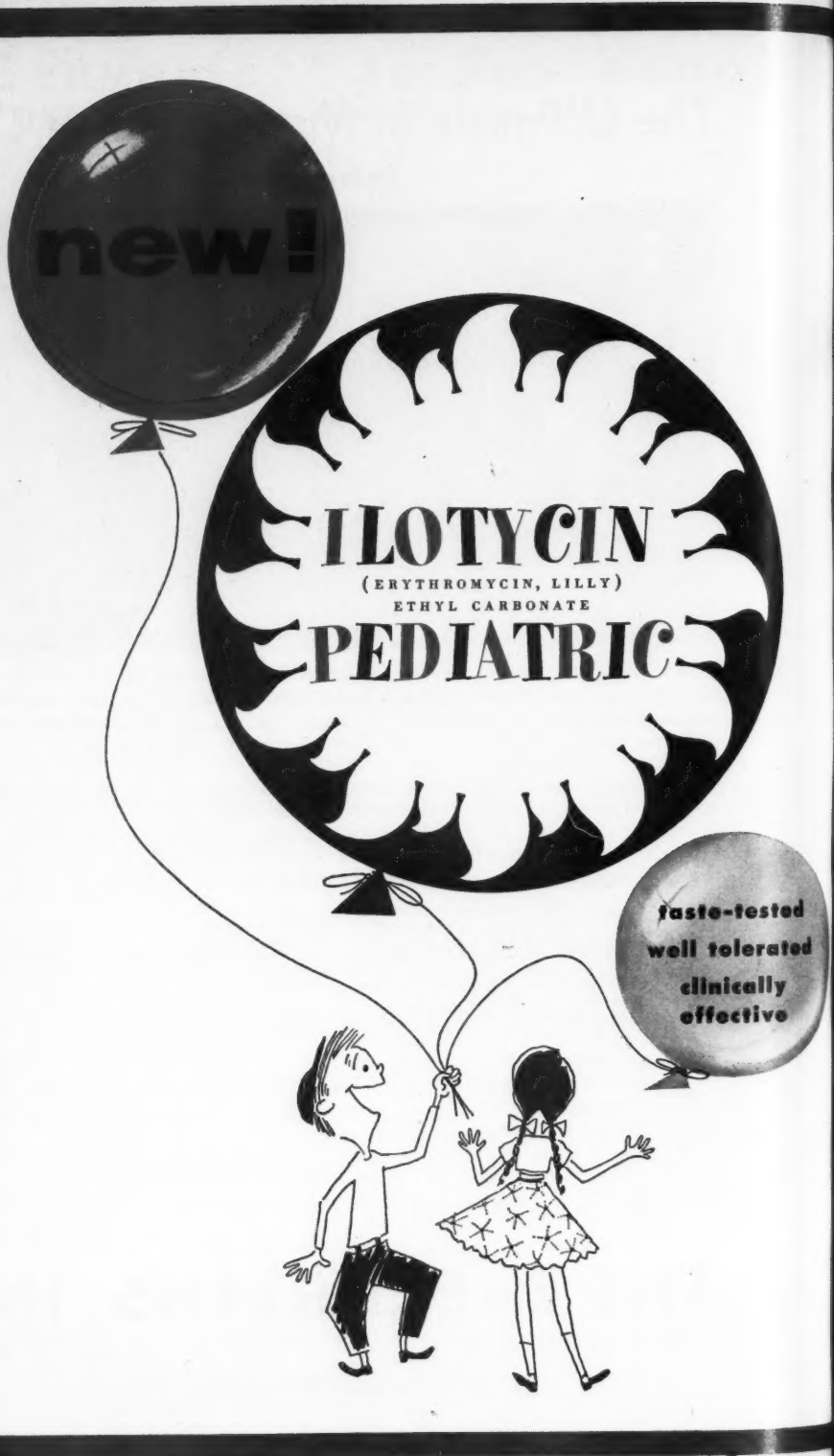
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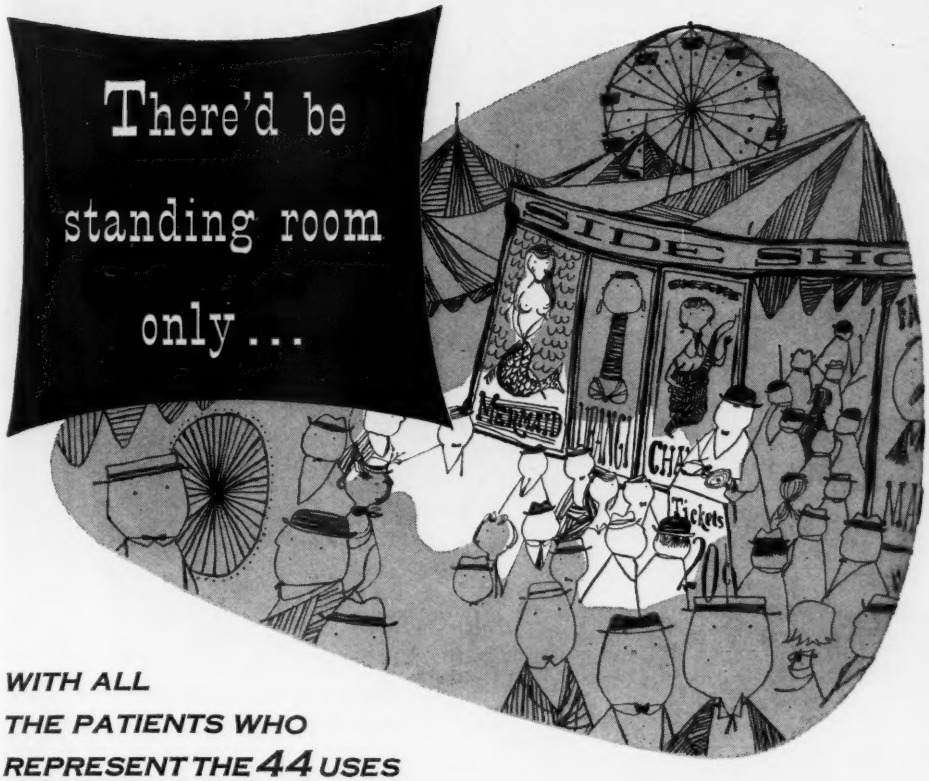

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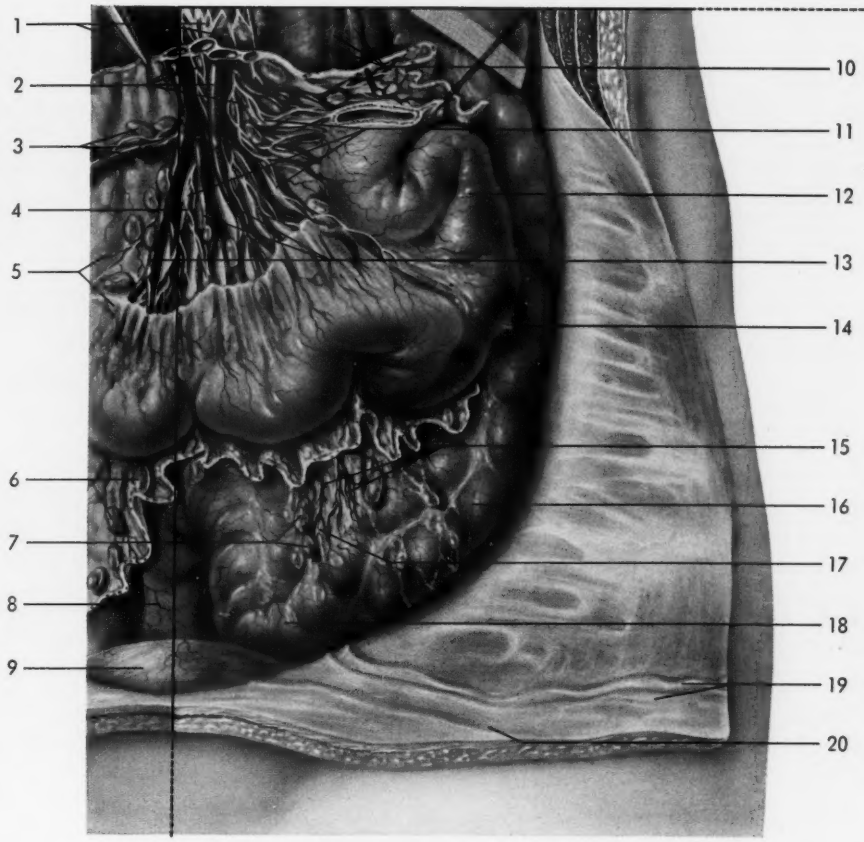

1 Vena cava, aorta and abdominal aortic plexus

2 Branches of superior mesenteric artery and vein

3 Ileocolic lymph node and ileocolic artery and vein

4 Sympathetic abdominal plexus

5 Mesentery

6 Mesenteric lymph nodes

7 Mesocolic lymph nodes

8 Rectum

9 Urinary bladder

10 Inferior mesenteric vein, left colic artery and ureter

11 Intestinal arteries

12 Ileum

13 Intestinal veins

14 Descending colon

15 Branches of sigmoid artery and vein

16 Iliac colon

17 Mesocolon

18 Sigmoid colon

19 Epigastric artery and vein

20 Lateral umbilical ligament

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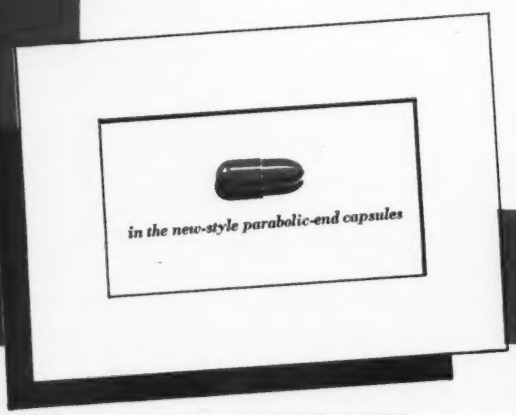

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Papilloma of the Thyroid: A Study of 50 Cases*

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Papillary tumors of the thyroid are the most common type of malignant goiter in this country. While they are very rare in mountainous goiter regions, they constitute between 30 and 64 per cent of the malignant goiters in lowland countries. From the standpoint of geographic pathology, it is of interest that in our material they originated 22 times in colloid goiters, in 7 instances in exophthalmic goiters, and 9 times in lymphadenoid goiters. These three types of goiter are also rare in mountainous goiter regions but very common in the lowlands.

While Woelfler[1] described the papilliferous adenoma and Wegelin[2] recognized a solid and cystic form of papilloma, neither related the clinical course of the various types of papilloma to their gross characteristics. The first to suggest this relationship was Graham,[3] in 1925. Moritz and Bayless,[4] in 1931, distinguished five forms of papilliferous tumors: (1) the papilliferous cystadenoma which they regarded as a benign hyperplasia of the lining epithelium of a cystic adenoma, (2) the papilliferous adenoma which represents epithelial proliferation within the follicles of a preexisting benign adenoma, (3) the papilliferous cystadenocarcinoma arising in a papilliferous cystadenoma, (4) the papilliferous malignant adenoma which shows a papillary intraacinar hyperplasia in a malignant adenoma, and (5) the papilliferous carcinoid which these authors considered as non-tumorous. Follow-up studies by Moritz and Bayless in papilliferous cystadenocarcinoma showed 8 recurrences in 14, but no death. Of their 8 patients with papilliferous malignant adenomas, 4 died. Moritz and Bayless concluded that only the cystic papillary tumors have a relatively benign course, while the solid papillomas behave like malignant tumors.

Our own experience does not confirm their conclusions. We found that cystic papillomas did not differ from solid forms; therefore, we classify all papillary tumors—as Crile[5] and Pemberton[6] do—as one group. Whether or not one is justified in calling all thyroid papillomas malignant, or potentially malignant, is of great importance to the patient, since the patient's entire future depends on this point of view. The patient may contemplate the purchase of new or additional life insurance. Should he be accepted, rejected, or "rated up" because he has a papilloma of the thyroid which his physician considers malignant? The executive who plans to expand his business may hesitate to do so, or he may stop future contracts or perhaps even retire, because he has been told that his papilloma of the thyroid is a malignant tumor. Should a young girl with a papilloma of the thyroid be subjected to total thyroidectomy and prophylactic bilateral radical neck dissection with resection of both sternocleido-mastoid muscles and of both jugular veins? And should she be warned against marriage or raising a family? How malignant is a papilloma of the thyroid, and what is the 5-year survival rate if only the involved thyroid lobe is removed?

Many pathologists are overly cautious and call all papillomas cancer, which of course is a great injustice to the patient. We should remember that of patients in whom malignancy of the thyroid is discovered only on microscopic examination, 86 per cent survive 5 years (R. C. Horn[7]); and we should not forget the advice given by such an experienced goiter surgeon as McGregor[8]: "When there is a difference of opinion between the surgeon based on gross appearance and

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TYPES OF SURGERY OF PAPILLARY ADENOCARCINOMA OBTAINED FROM LITERATURE

Clinic	*Lobectomy*	*Total*	*Radical Neck Dissection*	*Modified Neck Dissection*	*Lymph Node Groups*
1. University of Southern California Medical School		If nodes are not present, total thyroidectomy plus regional dissection is done.			If nodes are present, total thyroidectomy plus block dissection on involved side with resection of sternocleidomastoid muscle and jugular vein is done.
2. University of California School of Medicine, San Francisco	+		When indicated by metastatic glands		
3. Crile Clinic	+				Removal as nodes appear.
4. Hertzler Clinic	+				Removal as nodes appear.
5. Lahey Clinic	+		+		
6. Mayo Clinic	+		In presence of palpable nodes.		
7. Ochsner Clinic	+		+ In the presence of nodes		
8. Washington University Medical School and Barnes Hospital	+			Preservation of sternocleidomastoid muscle.	

different pathologists based upon microscopic study, no radical operation is advised. I have never regretted this course as yet."

The present report is based on a study of 50 patients with papilloma of the thyroid observed at the Hertzler Clinic during the years of 1940 to 1952, inclusive. They represent 31.2 per cent of our thyroid tumors.

Age and Sex: The papillomas were most frequent during the 4th and 5th decades of life. The medium age of our patients on admission was 41.8 years. Our youngest patient was 12 and the oldest 79 years of age. There were 45 women and 5 men in this series. Forty-five of the 50 patients had been aware of thyroid enlargement; in 5 patients the tumor was discovered on admission for various complaints. Twenty-four patients had known of their goiters for 1 year or less, while 8 were aware of neck enlargement for periods varying from 5 to 29 years. Six had noticed a marked increase in the size of their goiters during the last year.

Gross Pathology: Papillary carcinomas are usually unilateral. In 37 of the 50 specimens the papillary tumor was the only node, in 7 it had developed in a multinodular goiter. There were multiple tumors in 9 specimens, and 5 papillomas were bilateral. In 18 cases the nodes were sharply circumscribed, but in 22 the capsule was grossly invaded, and in 10 specimens the papillary tumor was without any capsule. Invasion of the neck muscles was noticed in 3 patients; the carotid sheath, trachea, and esophagus were involved

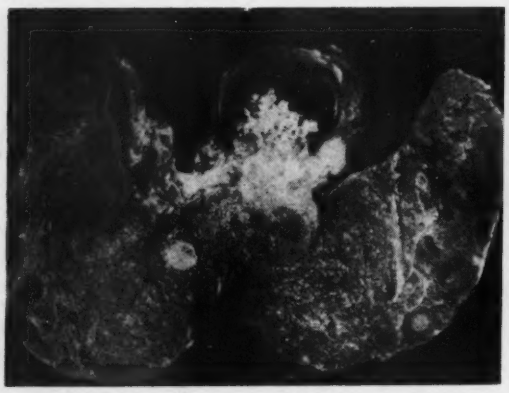

Figure 1. Cystic papilloma of thyroid isthmus from a 60-year-old woman. Weight of resected thyroid 48 grams. Patient died from hypertension one year after operation, without evidence of recurrence or metastasis.

Figure 2. Solid papilloma of right lobe of thyroid with "aberrant gland," in a 12-year-old boy. He now has a small tumor in right side of neck, 1 year after removal, and local removal is advised.

twice. Both of these patients were over 60 years old.

Papillary tumors are, as a rule, of small size. Twenty-one of our papillomas were less than 2 cm. in diameter, 15 measured between 2 and 4 cm., and only 7 tumors had a diameter over 6 cm. The largest tumor in our series measured 8 cm. The weight of the resected specimens in our cases varied from 12 to 156 grams. Twenty-five tumors appeared completely solid; 10 tumors had small clefts and lacunae filled with bloody or clear fluid. Distinctly cystic tumors with visible papillary projections were encountered in 15 cases, their contents being yellow or brown, and in 18 tumors gross hemorrhage was noticed. Microscopically the papillae varied in size and shape and were covered by cuboidal or columnar epithelium in one or more layers. Mitotic figures were infrequent. Calcium deposits were found in 13 of our cases and lymphocytic infiltration in 17.

While most of our specimens presented a papillary structure throughout, follicular and trabecular structures were not uncommon in the solid forms (Table I). Crile points out that it is of practical importance to distinguish these variants of papilloma from the more malignant non-papillary carcinomas. One of our patients, who had a large tumor which involved the wall of the esophagus, lived 5 years after incomplete operation in spite of a combination of a papilloma (grade 1), Hurthle cell tumor (grade 2), and solid carcinoma (grade 4).

We agree with Moritz and Bayless, that invasion of the thyroid tissue or adjacent structures and evidence of metastatic growth is the only criterion of malignancy. In only one tumor did we see blood vessel invasion. There was no strict correlation between histologic grade of malignancy and clinical course. We accept Willis'[9] standpoint that papillary adenomas cannot be distinguished cytologically from papillary carcinomas. Dunhill,[10] in his large experience, saw only two tumors which he regarded as "benign papillomas," and one of these later recurred. The discovery that an enucleated tumor has a papillary structure, however highly organized and quiescent in appearance, calls for lobectomy of the tumor-bearing lobe. We wait for paraffin sections from 4 to 5 blocks before making a histological diagnosis of malignant papilloma.

It may be of interest to surgeons who rely on frozen sections to know that even the pathologists of the Mayo Clinic, in spite of their unsurpassed experience in tissue diagnosis during operation, refuse to make a histologic diagnosis on papillary thyroid tumors from fresh frozen sections (Judd).[11]

The fact that Moritz and Bayless describe benign papilliferous tumors in aberrant cervical glands is further proof of the difficulty of diagnosing malignancy from the histologic structure, since there is general agreement today that the lateral aberrant thyroid tumors are metastatic extensions to the deep cervical lymph nodes from a primary papilloma of the thyroid.

Eight of our patients showed papillary tumors in cervical nodes on admission; 3 of these had had former operations.

The preoperative diagnosis in 42 of our 50 cases was benign goiter. Only in 8 patients was a malignant tumor suspected before operation.

Treatment: Bilateral subtotal thyroidectomy was performed in 18 patients and total thyroidectomy with radical neck dissection in two elderly patients. In 28 cases only one lobe was removed. In two cases removal of enlarged cervical nodes, and in two others resection of involved muscles, jugular vein, and wall of the esophagus, was done in addition to lobectomy. In 4 cases, so-called aberrant tumors were removed with thyroidectomy.

Follow-up: Of our 50 patients, 42 are known to be alive today, and 40 are without evidence of disease.

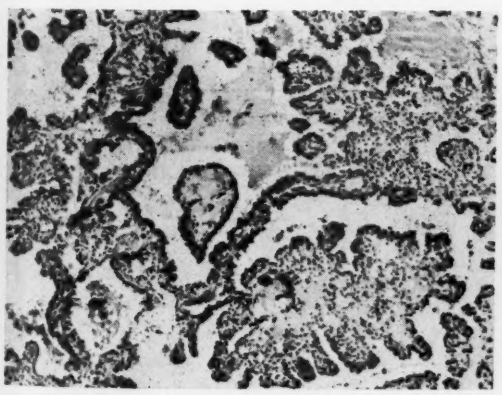

Figure 3. Microscopic section of "aberrant gland" of same case as Figure 2. The structure looks benign, in spite of being from a metastatic tumor.

Figure 4. Papilloma which had a solid appearance. Invasion of the capsule is evident, while cytologic criteria of malignancy are absent. No recurrence after bilateral thyroidectomy without neck dissection.

Case	SEX AND AGE			PATHOLOGY					METASTASES	
	Sex	*Age at onset*	*Age at diagnosis*	*Size of primary tumor*	*Capsule invasion*	*Solid papilloma*	*Cystic papillary*	*Histology*	*Cervical nodes*	*Mediastinal nodes*
1. 22629	M	31	36	6.5 cm.	—	+	—	Solid alveolar papillary	—	—
2. 23950	Fe	20	35	2.5 cm.	—	—	+	Papillary, large and small follicular	—	—
3. 23137	Fe	15	44	5 cm.	+	—	+	Solid alveolar papillary	—	—
4. 24391	Fe	50	54	3.0 cm.	Invasion fibrous tissue	—	+	Papillary and small follicular	—	—
5. 24449	Fe	34.5	39	1.3 cm.	Yes	—	—	Exophthalmic goiter	—	—
6. 24839	Fe	43	43	0.4 cm.	Yes—Thyroid tissue invas.	—	—	Trabecular, follicular papillary	—	—
7. 24985	M	27	30	6.0 cm.	Yes	—	+	Trabecular, follicular and papillary	—	—
8. 25247	M	50	50	1.0 cm.	No capsule. In thyroid tissue	—	—	Follicular papillary squamous cell metaplasia	—	—
9. 25359	Fe	27	39	1.1 cm.	No	—	+	Large and small follicular and papillary	—	—
10. 25699	Fe	22.5	23	1 x 2 cm.	No	—	—	Non-encapsulated infiltrating papilloma	—	—
11. 25912	Fe	42	43	7 mm.	Yes	—	—	Small follicular and papillary in exophthalmic	—	—
12. 25949	Fe	?	60	2.0 cm.	Yes	—	+	Papillary	—	—
13. 26125	Fe	60	61	1.5 cm.	No capsule	—	+	Papillary trabecular Hurthle cell	—	—
14. 26389	Fe	58	67	2.3 cm.	No capsule	—	—	Diffuse colloid trabecular and papillary	—	—
15. 26467	Fe	29	37	6.4 cm.	No	+	—	Papillary	—	—
16. 26500	Fe	22	23	3.0 cm.	No	—	—	Follicular and papillary	—	—
17. 26668	Fe	19	19	5.0 cm.	Yes	—	+	Follicular and papillary	—	—
18. 26726	Fe	51	51	5.0 cm.	Yes	—	—	Papillary	—	—
19. 26792	Fe	24	24	1.2 cm.	Yes	—	+	Follicular	+	—
20. 26927	M	28	45	1.1 cm.	No	—	—	Papillary and follicular	—	—
21. 26975	Fe	26	42	0.8 cm.	No	—	—	Large and small follicular and papillary	—	—
22. 27237	Fe	12	15	3.0 cm.	Yes	—	—	Follicular and papillary	—	—
23. 27546	Fe	56	60	2.5 cm.	Yes	—	—	Papillary	—	—
24. 27548	Fe	21	31	8.0 cm.	Yes	—	+	Small-follicular	—	—
25. 27643	Fe	67	79	6.0 cm.	Yes	—	—	Follicular, trabecular and papillary Hurthle cell	—	—
26. 27975	Fe	18	20	4.5 cm. to 1.0 cm.	Yes	—	—	Follicular and papillary	+	—

Case	TREATMENT						SURVIVAL (YEARS)		Remarks
	Subtotal bilat.	*Total*	*Lobectomy*	*Removal of lat. nodes*	*Radical neck dissection*	*Sterno-mast.; Jug. vein resect.*	*From time of onset*	*From time of diagnosis*	
1. 22629	−	−	+	−	−	−	10½ yrs.	5½ yrs.	At surgery there was diffuse enlargement of the right lobe. Living and well.
2. 23950	−	−	+	−	−	−	22 yrs.	7 yrs.	Solitary nodule palpable. Living and well.
3. 23137	+	−	−	−	−	−	36 yrs.	7 yrs.	Living and well. No recurrence.
4. 24391	+	−	−	−	−	−	8 yrs.	4 yrs.	Living and well.
5. 24449	+	−	−	−	−	−	12 yrs.	8 yrs.	Living and well.
6. 24839	−	−	Left lobectomy	−	−	−	6 yrs.	6 yrs.	Solitary walnut-sized nodule palpable in left lobe. Living and well.
7. 24985	−	−	Left lobectomy	−	−	−	10 yrs.	7 yrs.	Firm enlargement of left lower pole. Living and well.
8. 25247	−	−	Right lobectomy	−	−	−	5 yrs.	5 yrs.	Not encapsulated. Invaded surrounding thyroid tissue. Living and well.
9. 25359	−	−	Right lobectomy and isthmus	−	−	−	17 yrs.	5 yrs.	Recurrence?
10. 25699	−	−	Left lobectomy	−	−	−	7 yrs.	6.5 yrs.	Firm bilateral goiter.
11. 25912	−	−	1945—Left 1947—recurrence on left	−	−	1947—jugular vein and esophageal wall only	7 yrs.—5 yrs. from recurrence	7 yrs.	Not encapsulated. Primary surgery in 1945. Recurrence in 1947. Died in June, 1952.
12. 25949	+	−	−	−	−	−	1½ yrs.	1½ yrs.	Death 1½ years following surgery. Death from other causes.
13. 26125	+	−	−	−	−	Adherent—not invading	5 yrs.	4 yrs.	No capsule. Stuck to sternocleidomastoid, not invading. Clinically, goiter was fixed.
14. 26389	+	−	−	−	−	−	13 yrs.	4 yrs.	No capsule. In good health.
15. 26467	+	−	−	−	−	−	14 yrs.	6 yrs.	Living and well.
16. 26500	−	−	+	−	−	−	?	?	Unable to trace.
17. 26668	−	−	+	−	−	−	?	?	Unable to trace.
18. 26726	+	−	−	−	−	−	5 yrs.	5 yrs.	Living and well.
19. 26792	−	−	+	+	−	−	4 yrs.	4 yrs.	No recurrence.
20. 26927	+	−	−	−	−	−	22 yrs.	5 yrs.	Living and well.
21. 26975	−	−	+	−	−	−	?	?	Unable to trace.
22. 27237	+	−	−	−	−	−	7 yrs.	4 yrs.	Living and well.
23. 27546	+	−	−	−	−	−	4 yrs.	3 yrs.	Living and well.
24. 27548	−	−	+	−	−	−	13 yrs.	3 yrs.	Living and well.
25. 27643	−	−	+	−	+	Resection of jugular vein and piece of esophageal wall	17 yrs.	5 yrs.	Died five years after operation.
26. 27975	−	−	+	−	−	−	4 yrs.	2 yrs.	Two years living and well. Biopsy on 4/22/48. Lobectomy on 4/28/53.

Case	SEX AND AGE			PATHOLOGY					METASTASES	
	Sex	*Age at onset*	*Age at diagnosis*	*Size of primary tumor*	*Capsule invasion*	*Solid papilloma*	*Cystic papillary*	*Histology*	*Cervical nodes*	*Mediastinal nodes*
27. 28002	Fe	4	24	1.2 cm.	Yes	—	—	Follicular and papillary	+	—
28. 28307	Fe	45 ?	50	0.4 cm.	No	—	—	Papillary with tissue invasion	—	—
29. 28979	Fe	?	47	1.7 cm.	No	—	—	Follicular, trabecular and papillary	—	—
30. 29195	Fe	?	50—Found on routine Px.	1.0 cm.	No	+	—	Papillary	—	—
31. 29218	Fe	?	67—Found on routine Px.	0.8 cm.	No	—	—	Papillary	—	—
32. 29331	Fe	22	22	1.4 cm.	No	—	—	Follicular and papillary	—	—
33. 29706	Fe	43	43	1.5 cm.	Yes	—	—	Papillary on right and left	—	—
34. 29861	Fe	48 ?	48	0.6 cm.	No	—	+	Follicular and papillary	—	—
35. 30117	Fe	57	59	6 cm.	No capsule	—	—	Follicular and papillary	—	—
36. 32785	M	8	12	4 cm. Three metastases, 1.8 to 0.8 cm.	Yes	+	+	Trabecular and papillary	+	—
37. 23786	M	30	34	4.0 cm.	Yes	—	—	Follicular and papillary	—	—
38. 24114	Fe	35	52	6.0 cm.	No	—	+	Papillary	+	+
39. 23696	Fe	35	41	5.0 cm.	No	—	—	Trabecular, follicular and papillary	—	—
40. 23293	M	64	64	3.5 cm.	No	—	—	Follicular and papillary	—	—
41. 20804	Fe	50	50	1.2 cm.	Yes—invasion of thyroid tissue	—	—	Papillary	—	—
42. 26053	Fe	29	29	1.1 cm.	Yes	—	—	Follicular and papillary	—	—
43. 21022	Fe	54.5	56	6.0 cm.	Yes—Grossly	—	+	Trabecular, follicular and papillary	—	—
44. 25169	Fe	33	34	8 mm.	Yes	—	—	Papillary	—	—
45. 20852	M	33	36	2.0 cm.	Yes	—	—	Papillary	—	—
46. 28281	Fe	35	38	4.5 cm.	Yes	—	+	Trabecular, follicular and papillary	—	—
47. 21490	Fe	25	25	2.5 cm.	Yes	—	—	Papillary	—	—
48. 33372	Fe	31	34	4.5 cm.	Yes	—	—	Trabecular, follicular and papillary	—	—
49. 22837	Fe	26	28	6.0 cm.	No	—	—	Follicular and papillary		
50. 23423	Fe	46	46	8 mm.	No	—	—	Trabecular, follicular and papillary	—	—

Case	TREATMENT						SURVIVAL (YEARS)		Remarks
	Subtotal bilat.	*Total*	*Lobectomy*	*Removal of lat. nodes*	*Radical neck dissection*	*Sterno-mast.; Jug. vein resect.*	*From time of onset*	*From time of diagnosis*	
27. 28002	—	—	+ Homolateral node resection	+ Done here	Bilateral since last seen here	—	24 yrs.	4+ yrs.	Patient had eight operations elsewhere between 4 and 24 years. Two operations here. Living and well. X-ray therapy elsewhere since surgery here. Negative chest X-ray in July, 1950.
28. 28307	+	—	—	—	—	—	8+ yrs.	3+ yrs.	Living and well.
29. 28979	+	—	—	—	—	—	3 yrs.	3 yrs.	Living and well.
30. 29195	—	—	+	—	—	—	3 yrs.	3 yrs.	Living and well.
31. 29218	—	—	+	—	—	—	2 yrs.	2 yrs.	Living and well.
32. 29331	—	—	+	—	—	—	1 yr.	1 yr.	Living and well.
33. 29706	+	—	—	—	—	—	?	?	Unable to trace.
34. 29861	—	—	+	—	—	—	2 yrs.	2 yrs.	Living and well.
35. 30117	—	+	—	—	+ Left side only	+ Left side only	5 yrs.	2 yrs.	Living and well. Muscle invasion at surgery.
36. 32785	—	—	+	—	—	—	4½ yrs.	½ yr.	Postoperative X-ray therapy. Living with recurrence. To have radical neck dissection.
37. 23786	—	—	+	—	—	—	6½ yrs.	2½ yrs.	Living and well.
38. 24114	+	—	—	+	—	—	3 yrs.	6 yrs.	3 Operations: (1) Right lobectomy in 1944. Muscle invasion with direct extension to upper mediastinum. (2) Removal of three recurrent nodes, 1948. (3) Metastasis in neck muscle, 1949. Patient died 6 years following surgery with pulmonary metastases, 1950.
39. 23696	—	—	+	—	—	—	15½ yrs.	9½ yrs.	Living and well. No recurrence.
40. 23293	—	—	+	—	—	—	10 yrs.	10 yrs.	Living, in poor health. No recurrence.
41. 20804	+	—	—	—	—	—	1 yr.	1 yr.	Living and well.
42. 26053	—	—	Bilateral lobectomy in 1946	+ 1946	—	—	11 yrs.	6 yrs.	First surgery in 1941 elsewhere. Recurrent symptoms in 1943 and again in 1946, with surgery in 1946. Living and well 6 years since last surgery in 1946.
43. 21022	—	—	+	—	—	—	11.5 yrs.	10 yrs.	Living and well. No recurrence.
44. 25169	+	—	—	—	—	—	8 yrs.	7 yrs.	Living and well. No recurrence.
45. 20852	+	—	—	—	—	—	13 yrs.	10 yrs.	Living and well. No recurrence.
46. 28281	—	—	+	—	—	—	7 yrs.	4 yrs.	Living and well. No recurrence.
47. 21490	+ 1941	—	—	—	—	—	12 yrs.	12 yrs.	Living and well. No recurrence.
48. 33372	—	—	+	—	—	—	6 yrs.	3 yrs.	Living and well.
49. 22837								8 yrs.	Living and well.
50. 23423	+ 1930	—	+ 1943	—	—	—	10 yrs.	10 yrs.	Surgery in 1930—Hyperplastic goiter. Surgery in 1943—Papillary adenocarcinoma. Living and well 10 years.

Acknowledgment: We are indebted to V. E. Chesky, M.D., for the use of 33 cases from his service, and to I. A. Koeneke, M.D., for the use of 12 of her cases.

Three patients are dead, and 5 are lost in the follow-up study. One died at the age of 58, 6 years after her first operation which included radical neck dissection. One patient died at 84 years of age, 5 years after incomplete removal of a papillary carcinoma which had involved the esophagus and the deep jugular vein. The third patient died from hypertension at the age of 60, one year after thyroidectomy, without showing signs of recurrence or metastasis.

Characteristic of the long life history of most papillomas is the case of a 26-year-old woman who had had 8 neck operations before she came to our clinic with a large cervical node. After its removal another "aberrant thyroid" appeared. Following total thyroidectomy, which revealed a primary papilloma in the homolateral lobe, she is now free of symptoms 22 years after her first operation. Another patient, with lateral papillary tumors, had 3 operations for enlarged cervical lymph nodes and is now free of symptoms 7 years after her last operation. We have followed 23 of our patients for more than 5 years; 18 are known to be alive and free of disease, while two have had recurrences, and 2 are dead of their thyroid disease. The over-all 5-year survival rate is 87 per cent.

CONCLUSIONS

Papillary tumors are the most common form of tumors of the thyroid. Solid papillomas are not more malignant than cystic forms, but have, even in combination with follicular and trabecular structures, a relatively benign course. Invasion of the capsule or the presence of metastasis is the only criterion of malignancy of a papillomatous tumor.

So-called "aberrant thyroids" are metastases of a primary tumor in the thyroid and will continue to recur as long as the latter is not removed.

Since papilloma of the thyroid is a relatively benign tumor, we regard prophylactic radical neck dissection as not justified. When the papilloma is confined to the thyroid, no recurrence or death is to be feared following removal of the tumor-bearing lobe. With this conservative operation our over-all 5-year survival rate was 87 per cent. Since patients with papilloma of the thyroid are often young women, prophylactic radical neck dissection in every case of papilloma of the thyroid seems to us an unnecessary mutilation. We feel that the diagnosis "cancer" is a great injustice to a young patient who has more than a 90 per cent chance of cure after simple lobectomy. We prefer therefore to call this tumor papilloma of the thyroid, as is generally done in European countries.

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Gynecological Pain*

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The present day practitioner of medicine considers the patient as an organism with many systems, some of which are definitely inter-related. The gynecologist does not consider the female as having a genital tract entirely divorced from other regions. He realizes that each individual has many systems and that disease of other systems can directly or indirectly influence the genital tract. He also remembers that pelvic pathology is not always the sole cause of many symptoms which she may present. There are many individuals who may present familial and/or hereditary characteristics or developmental anomalies. These must always be considered in evaluating one's findings.

Pain is a symptom which is common to all systems of the body. The genital tract is, of course, no exception in this respect. Pain is the most common complaint given to a gynecologist as a reason for an examination. Pelvic pain accounts for at least 35 per cent of the admissions to a gynecological ward. Unfortunately, it is too often customary to regard all feminine pain located anatomically between the umbilicus and the perineum as originating in the generative organs, especially if it is associated with or exaggerated by menstruation. It matters little if the

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pelvic organs are found to be normal, for the idea is firmly fixed that pelvic pain is always genital in origin. There is also an unfortunate tendency at times to invent pelvic pathology such as a cystic or prolapsed ovary, or even a displaced uterus, in order to fit the patient's symptoms. In a desire to relieve pelvic pain, many operations are done in the name of gynecology.

Diddle, O'Connor and Winebrenner,[1] reporting recently on unnecessary gynecological operative procedures in five non-teaching hospitals, found 28 per cent of 889 operations were unjustified. Their opinion was based on a correlation of clinical indications and histopathology. They were also of the opinion that the great majority of 155 uterine suspensions and 75 ovarian resections were unjustified. The principal complaints given by the patients were abdominal pain or backache. They concluded that the majority of hysterectomies done for such reasons were unjustified as tissues removed were normal. These women most probably had psychosomatic or orthopedic problems. They were also of the opinion that such conditions existed in similar hospitals throughout the entire country.

Norman F. Miller,[2] in a paper entitled "Hysterectomy, a Therapeutic Necessity or Surgical Racket," found that in 22 per cent of the cases, the main symptom and often the only one was lower abdominal pain.

At a meeting of the Southern Gynecological and Obstetrical Society in Dallas in 1951, William F. Guerriero[3] gave a presentation of gynecic-like pain. Of 1,100 patients whose main complaint was pelvic pain, he found 42 per cent had no pelvic pathology. These patients were grouped as follows:

Urologic: Forty-four per cent, of which 75 per cent were found to have pain as a result of posterior urethritis, a condition described by his group as the female prostate.

Orthopedic: Twenty-five per cent, mostly due to postural and mechanical conditions.

Medical: Seven per cent. Among the medical conditions most commonly found responsible for gynecic-like pain were:

Colitis in 40 patients.

Diverticulitis in 8 patients; (must always be considered in women over 40).

Amebiasis in 5.

Cancer of the large bowel in 3.

Polyps of the gastrointestinal tract in 4.

The remaining 24 per cent were classified as a mixed group and included conditions in other systems than the ones already mentioned. It is of special interest to note that 12 per cent of the mixed group were found to be of psychic origin.

James C. Doyle,[4] in reporting in 1953 on unnecessary hysterectomies, found abdominal pain the only complaint in 185 patients and backache the single symptom in 86 others subjected to hysterectomy, in none of whom did a pathological lesion actually exist.

When the consultant gynecologist finally sees these patients, they often have had many surgical procedures, including appendectomy, removal of part or all of an ovary, or uterine suspension, and, unfortunately, they still have lower abdominal or pelvic pain.

The uterus, tubes, and ovaries are innervated from the autonomic nervous sytem, and sensations from them are produced in the same fashion as visceral sensations from the intestine, liver, or spleen. The parietal peritoneum, vagina, and external pudendal regions are innervated by myelinated fibers, and pain sensations originate directly from these areas. Pelvic pain as classified by Mengert[5] can be of genital or extragenital origin as follows:

I. *Pain of Genital Origin:*

1. Gonorrhea, pelvic inflammatory disease, pelvic cellulitis, hemorrhage.
2. Uterine prolapse, adhesions, twisted pedicle of ovarian cyst.
3. Periodic distention of endometrial implant.
4. Tumor incarcerated in pelvis.
5. Rupture of uterus, tube, or bladder.

II. *Pain of Extragenital Origin:*

1. Pelvic Neurosis.
2. Pain originating in other pelvic structures.
   a. Bony, sacro-iliac.
   b. Urinary tract.
   c. Intestinal tract, colitis, diverticulitis, appendicitis.

Time prevents my discussing each one of these conditions in detail, so I will confine my remarks to those disorders that are commonly encountered and at times confused. Among the acute conditions that must be considered are appendicitis, acute salpingitis, extra uterine pregnancy, twisted pedicle of an ovarian cyst, ovarian hemorrhage and ureteral calculus.

Acute appendicitis is perhaps the most frequent cause of lower right quadrant pain. A typical case with sudden onset of pain which is at first umbilical or epigastric, and later localized near McBurney's point with gastrointestinal disturbances, usually offers no diagnostic problem. Where this does not occur, one must consider, among other acute conditions, salpingitis. This differentiation should not be difficult in the average case. The history of exposure, the onset of symptoms with high fever, chills, high leukocyte count, and bilateral abdominal pain are factors that seldom occur in other conditions. Ruptured tubal pregnancy must always be considered, and in such a condition the history will be of great value. Where rupture has taken place, as evidenced by hemorrhagic shock, one should have no difficulty in determining

this. Cul-de-sac puncture is a valuable aid in such a diagnosis.

Hemorrhage from a ruptured follicle or corpus luteal cyst or ovulation may at times be confusing. A ruptured graafian follicle with severe pain at ovulation is distinguished by its timing and recurrence. There have been many laparotomies done for supposed acute appendicitis, where ovarian hemorrhage had occurred.

Differential diagnosis is at times difficult. During the past six months I have seen two 14-year old girls with acute abdomens where appendicitis was the most likely diagnosis but where paraovarian cysts with hemorrhage into the mesosalpinx were found to be the primary condition.

Twisted pedicle of an ovarian cyst usually presents a clinical picture of acute abdomen. The main symptom, of course, is abdominal pain of severe type. Severity of shock depends on the size of the cyst and amount of disturbed blood supply. Diagnosis is easy if one is aware of the pre-existence of such a cyst prior to the acute attack.

Among acute conditions of extragenital origin, ureteral colic often causes pelvic pain, and one should always rule it out before surgery is performed. Examination of the urine with findings of red cells and x-ray of the kidney and bladder region will usually give sufficient information.

A correct diagnosis of those conditions that cause an acute abdomen can, in the average case, be successfully made. As the great majority of these conditions require immediate surgery, this differentiation is not always obligatory. It is often made only at the time of surgery. Most gynecologists, however, prefer to do an occasional laparotomy needlessly rather than to fail to do so and later have a ruptured extrauterine pregnancy or ruptured tubal abscess found at autopsy.

Chronic pelvic pain or lower abdominal pain may be the result of a multitude of conditions. One must always rule out pain of bony origin before holding the pelvic organs responsible. H. C. Pitkin,[6] in discussing orthopedic causes of pelvic pain stated, "Pelvic pain which regularly accompanies physiologic relaxation of the female pelvis that precedes menstruation, and is thought to be a hormonal disturbance, is so common that many women accept it as being unavoidable. Relaxation of the intrapelvic joints is one of the commonest causes of pelvic pain in women. Other causes are disturbed function of the pelvic and lumbar joints, muscles and ligaments, such as that caused by sprains or strains. Pelvic pain can also result from structural changes in the pelvic and lumbar spines such as that caused by congenital defects, fractures, degeneration of intervertebral discs, arthritis, or neoplasm. Disturbed function of the lower extremities such as flat feet can be responsible for pelvic pain."

When pressure on tender areas of the trunk or lower extremity produces or aggravates referred pain, and when procaine injection relieves this pain, one may assume that the site of origin has been located. In inaccessible areas, an estimate of articular function is necessary to locate the origin of pain. There is no question that the orthopedists have at times relieved pelvic pain after the gynecologists or urologists have been unable to do so. Complete comprehension of such cases has often been narrowed because of over-specialization.

Chronic pelvic pain is often the result of cervical pathology. Collins, Schneider and Baggs,[9] in reporting on benign lesions of the cervix, found that pelvic pain was the chief complaint in 50 per cent of 226 cervical stumps. Although lower abdominal pain and backache are caused by disease of intraabdominal organs, these symptoms are frequently the result of a chronically diseased cervix. Sir James Young,[10] in 1930, first described this important relation between lower abdominal pain and a chronically diseased cervix, and called attention to the diagnostic significance of a clinical procedure for reproducing the patient's symptoms.

On the Tulane service, the following clinical test has proved of great help in determining the part the cervix or cervical stump plays in producing the patient's symptoms. In the absence of fixed pelvic masses, the ability to reproduce a patient's symptoms on motion of the cervix means that the cervix is the etiological factor. The important factor is reproduction of pain and not merely production of pain. Production of pain is possible in any woman if the examination is vigorous. Reproduction of pain means the ability on gentle palpation to bring about the same pain in the same locality, with the same distribution, as complained of by the patient in her history. This is true whether the patient complains of lower abdominal pain, backache, dyspareunia, and bearing down sensation, alone or in any combination.

The examining fingers should first be inserted into one lateral fornix and the cervix pushed to the opposite side of the pelvis. If the pain is on the right side, the cervix should be pushed to the left, with resulting tension on the right cardinal ligament and vice versa. The patient should then be asked, "Is this the same pain of which you complain?" —not merely, "Does this hurt?" It is possible to displace a healthy cervix considerably without discomfort. If backache is a complaint, the examining fingers are introduced into the posterior fornix, the cervix is pushed toward the symphysis pubis, and the uterosacral ligaments are placed under tension. Traction on the infected cervix, where ligamentous involvement is present, will also reproduce lower abdominal pain or backache. Reproduction of the patient's backache means that the cervix is the causative factor and that

appropriate methods of therapy directed toward curing the chronic cervicitis will produce relief.

Deep seated dyspareunia is frequently due to a chronically diseased cervix. Whether or not the cervix is the cause of dyspareunia can be decided by motion of the cervix and attempting to reproduce the same type of pain as the patient experienced during coitus. Removal of the diseased cervical stump is usually followed by complete relief.

The symptoms initiated and maintained by chronic cervicitis are due to lymphatic extension of the responsible organism or organisms. Extension down the lymphatics of the uterosacral ligament produces backache. Extension laterally into the lymphatics of the broad ligament produces low abdominal pain. Treatment consists of cauterization, conization, or complete excision of the cervical stump, depending upon the extent of the pathology.

Uterine prolapse of a second or third degree can at times be held responsible for some instances of chronic lower abdominal pain, and this can be corrected by procedures depending upon the age of the individual. In the young individual with considerable prolapse, abdominal suspension and repair of the pelvic relaxation is occasionally necessary. This, however, should always be preceded by the use of a pessary as a therapeutic test. In women in their late 30's and over, any prolapse of the uterus should be treated by a vaginal hysterectomy.

A retrodisplaced uterus used to be thought of as an important factor in the cause of chronic pelvic pain. The present day gynecologist, however, does not recognize this. There are many women with retrodisplaced uteri who do not ever have chronic abdominal pain or backache. Subjecting such individuals to uterine suspensions is unfortunately an error that is too often committed.

Chronic pelvic pain is a symptom that often accompanies chronic pelvic inflammatory disease and endometriosis. This pain varies in intensity, depending upon the extent of the inflammatory lesion or the extent of the endometrial process. Total abdominal hysterectomy and bilateral adnexectomy is the procedure advocated in those patients having extensive pelvic inflammatory disease that has caused severe pain and menstrual disturbances. The same procedure is advisable in patients having extensive endometriosis. Fortunately, however, there are many patients having endometriosis of such a degree that it does not warrant a radical procedure.

Pelvic sympathectomy is at times recommended for the relief of severe primary dysmenorrhea that has not responded to any conservative treatment. It is also used in endometriosis where conservative surgery can be performed but the possibility of dysmenorrhea continuing is great, in instances where abdominal surgery is performed for cancer of the uterus, tubes, or ovaries, and in some instances where repeated laparotomies have failed to relieve pain of unknown origin.

It is difficult to separate real pain from psychic pain, or that which is expected to occur when bleeding appears. It is necessary to differentiate between these two in order to select properly the patient that will be helped by sympathectomy. Sturgis[8] and Allbright, and later Sturgis and Meigs, found a test satisfactory in 86 to 90 per cent of patients operated upon for relief of dysmenorrhea. They proved that essential dysmenorrhea could be relieved by stilbestrol or estradiol dipropionate, given in proper dosage to prevent ovulation, it being well known that anovulatory cycles are painless. This is a test to separate real dysmenorrhea from psychic dysmenorrhea. To prevent ovulation, begin on the first day of the menstrual cycle with 1 mg. of stilbestrol daily for 20 days. If a flow occurs three to five days later, it is considered anovulatory and it should be painless. In only those patients who have no pain at such a period should a resection of the hypogastric sympathetic nerves be done.

Phaneuf[7] reported the results in 76 patients between the ages of 13 and 38 on whom presacral neurectomy had been done for relief of intractable dysmenorrhea, where all simpler methods of treatment had failed. Of 68 on whom a follow-up was made, 40, or 59 per cent, had satisfactory results; that is, pain was absent or barely noticeable after surgery. Nineteen, or 29 per cent, were improved; that is, they had pain but considered themselves greatly relieved. Eight, or 12 per cent, were unimproved.

In patients who are properly selected and on whom a correct hypogastric sympathectomy is performed, clearing out all the nerve fibers in the interiliac trigone down to the periosteum, there should be universally good results. The poor results seen are in badly selected cases or because of inadequate nerve resection.

Pelvic neurosis is one of the extragenital causes of pelvic pain. It often originates from sexual incompatibility, infidelity, sterility, and other sexual factors that are the basic aspects of a woman's life. It is always difficult, and at times impossible, to obtain such information from the patient. Only by careful examination, eliminating abnormal pelvic organs, can one even suspect such a condition as pelvic neurosis. Even then, however, careful and patient questioning must be carried on before one can hope to arrive at a diagnosis. When this has been done, such a patient no longer should remain under the care of a gynecologist but should be referred to the psychiatrist. Too often, however, such individuals have been subjected to one or more pelvic operations before a diagnosis of pelvic neurosis has been established. Fortunately, most individuals are quite stable. Others who have low pain thresholds or neurotic dispositions are less stable, and they are thus likely to over-react to even

slight lesions of pain-sensitive structures. In persons of this latter group there often develops over-tenseness of the muscles, especially those in the low back. Anxiety concerning menstruation or sex factors, in addition to perhaps poor posture and poor nutrition, leads to perception of pain, the misinterpretation of which, under neurotic pressure tendencies, increases their anxiety and brings about a vicious circle of pain, anxiety, and increased tension.

Under different conditions, however, these same individuals may evidence their neurotic tendencies by cardiac neuroses with chest pain or gastric neuroses with upper abdominal pain and gastrointestinal disturbances. Most psychiatrists will admit that they are unable to offer a satisfactory explanation as to why some patients exhibit pelvic neuroses and others cardiac or gastric. Surely the gynecologist or general practitioner should not be criticized when he is unable to cope with such patients.

One must try to reach a happy medium in dealing with a woman whose chief complaint is lower abdominal pain. At times the gynecologist must be quite conservative and at other times appear to be radical. One's experience is often the only measure that can be used in determining which course to follow. The internist is often the first to see such a patient, and too often he considers it as a strictly medical condition. Also too often he fails to consider the pelvic organs as a possible cause and even fails to do a pelvic examination. He attempts to fit some disease pattern to the symptoms complained of. I cannot too strongly stress the importance of pelvic examination as routine in any woman. This is especially true, of course, in any woman complaining of pelvic pain. If this is not done by the internist, he should then have a gynecological consultant as a part of his routine examination of such an individual.

Rectal examination is often a valuable aid for diagnosis of pelvic pathology where the main complaint is pelvic pain. This examination is of considerable value in young females with an intact hymen. Often it is the only means of determining pelvic pathology in such patients. This is especially true in uterosacral endometriosis and in pelvic infections with fixation of the pelvic structures in the cul-de-sac. Such a procedure should always be a part of the gynecologist's examination, just as it should be part of the internist's.

It is, of course, quite true that often the surgeon is at fault and surgical procedures are done without adequate findings. Too many times are normal ovaries removed for simple cysts or suspended because of questionable prolapse. Too often are uteri suspended because of retroversions that are not the cause of pelvic pain. Too often are normal uteri removed where pelvic pain has been the only complaint. In most instances, this pain remains after such surgery is done. When the consultant gynecologist finally sees the unfortunate patient, she has a battle scarred abdomen and still has her pain. Such cases are, of course, the exception, but I can assure you they do exist.

CONCLUSIONS

What then can we do about lower abdominal or pelvic pain? The differential diagnosis of the acute abdomen can usually be successfully carried out. As most acute conditions require immediate surgery, the positive differentiation is not necessarily obligatory. Chronic pelvic pain presents a more difficult situation, and differentiation at times is almost impossible. Thorough examination from an orthopedic standpoint should always be done. By such careful examination, orthopedic causes can usually be ruled out.

It is difficult to make a diagnosis of pelvic neurosis. Sexual incompatibility, infidelity, or even sterility may be causal factors that at times are difficult to evaluate. As a matter of fact, the presence of these factors is most difficult to obtain from the patient.

We cannot always be sure whether pain is of genital or extragenital origin. We can, however, usually differentiate between anatomically normal and abnormal pelvic organs. We can refuse to operate except where definite disease entities exist. The only exception is exploration in the presence of prolonged and persistent pain of obscure origin. Unfortunately, even in such cases, we will in the majority find no pathology.

Presacral sympathectomy must be reserved for those cases that have been carefully selected and are proven to be true primary dysmenorrhea, or at times in endometriosis where conservative surgery can be done but some continued pelvic pain can be expected.

Where no pathology is found on examination of patients complaining of chronic pelvic pain, we must not do meddlesome surgery, for if we do we must remember that such patients will still have pain.

The internist can help by always including as part of his physical examination a pelvic and rectal examination. If he does not feel himself qualified to interpret any abnormal findings, then he should refer his patient for proper pelvic examination and diagnosis.

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The Role of Intervertebral Disc in Backache

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I have only two points to make in this paper. First, pathology in the intervertebral disc causes 75 per cent of orthopedic backaches; and second, the history in these cases is sufficient to make a diagnosis and to indicate treatment. I believe that emphasis of these two conclusions will materially aid our interpretation of the complaint, prevent our wandering in a therapeutic labyrinth, and save us the embarrassment of having our patients stray to more mechanically and less scientifically trained practitioners.

It is easily understandable why we physicians have had a muddled concept of backache when we realize that the pathological basis for an adequate understanding of it was promulgated only 15 to 20 years ago by Orthopedist Barr and Neurosurgeon Mixter of Boston. Previous to that time, every specialty accepted backache as its province, and it was treated by the dentist with tooth extraction, by the E.N.T. department with tonsillectomy, by the internist with sodium salicylate, by the urologist with kidney suspension and prostatic massage, by the general surgeon with hemorrhoidectomy, by the psychiatrist (probably effectively) by the daily hour's rest on a couch, and by the gynecologist with uterine suspension. If the patient ever got to an orthopedist, he was treated with arch supports just as effectively as he was treated by all the other branches of medicine.

The role of the intervertebral disc, gradually clarifying, has converged our attention to the seat of the pathology and has dispelled previous confusion of our attack on the symptom. Barr and Mixter, in their original paper, attempted only to explain the pathology of some of the cases of sciatic pain. In describing what they thought to be the pathology of an occasional case of sciatic pain, they not only explained most cases of sciatic pain but also most cases of low back pain.

We recently analyzed a large group of patients complaining of low back pain. Competent physicians had previously eliminated from this group possible extraneous pathology, and these patients were referred to us as orthopedic problems. To be concise, and as an aid to memory, the percentages which follow are approximate and are reduced to round figures.

Seventy-five per cent of these orthopedic backaches we diagnosed as secondary to mechanical-degenerative pathology of the intervertebral disc. Fifteen per cent were due to mechanical abnormalities which were congenital or developmental, as spondylolysis, spondylolisthesis, transitional vertebrae, vertebral osteochondrosis, and other less frequent anomalies. The remaining 10 per cent were due to disease processes in the spine or its motors such as infections, new growth, arthritis, myositis, fibrositis, etc. As you can see from our findings, arthritis has lost its pre-eminent position and has receded to a minor role in diagnosis of low back pain.

Because the intervertebral disc is the offender in so large a proportion of patients complaining of low back pain, it deserves careful study, consideration, and understanding. The understanding of disc pathology makes comprehensible many previously mysterious incidents such as the instantaneous onset of symptoms, the frequently substantiated good results of manipulation, the symptomless intervals between attacks, the relief with recumbency, and the location and character of sciatic pain. When the pathology of the intervertebral disc is well understood, the unity of the syndrome is apparent, and sciatic pain is seen to designate only the site of pathology rather than a difference in pathology. Sciatic pain and backache are in these cases manifestations of the identical pathological process.

The primary functions of a disc are to support weight and to allow movement between the bodies of two adjacent vertebrae. To so function the disc is composed of two parts, the nucleus and the annulus. The nucleus lies near the center of the disc and, as a mass of semiliquid material, being incompressible, bears the superimposed body weight. The annulus surrounds the nucleus and is composed of concentric rings of fibrous tissues. These rings, being compressible and elastic, allow movement. The nucleus and the annulus combined form a machine which functions as a hydraulic ball bearing. The machine works because the annulus, being compressible and elastic about the circumference of the disc, allows movement between the two vertebral bodies while confining the nucleus in the center of the disc to support weight.

The normal disc, in transverse section, can be compared to the cut surface of an onion with the rings of the annulus corresponding to the layers

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of the onion. These rings are regular with fairly distinct outlines, gradually losing their detail as they merge centrally into the nucleus. In an adult degenerated disc, however, the appearance is quite changed and, instead of looking like a section of an onion, looks more like a mass of combed wet paper. The rings of the annulus lose their distinctness and pattern and lie as friable, wavy, irregular, incomplete bands without precision in structure. The nucleus also changes in appearance as it loses some of its moisture.

It is interesting and important to consider the blood supply of the disc in relation to its early degeneration. In infancy the disc is supplied by vessels passing from the cancellous vertebral bodies through the cartilage plates into the disc tissue. This blood supply is obliterated gradually over a period of years, and by the age of 20 completely disappears. The disc thereafter is dependent upon diffusion of the body fluids from contiguous areas for its nutrition. With this inadequate nutritive supply, the degenerative processes are inevitable and occur both in the annulus and in the cartilage plates. With progressive degeneration the annulus loses its elasticity, and the loss of its elasticity seals its fate, and it lies unprotected by its normal resilience, awaiting its rupture by any sudden force or by the constant erosion of normal functional strain.

It is impossible to describe exactly the mechanics of the involved disc in the course of an episode, but pathological specimens and operative findings may be correlated with the symptoms to deduce a reasonable sequence of events. The friable annulus gives way at some point in its circumference, weakening its ability to maintain the nucleus in its central position. The nucleus is propelled into the crevice or bulge in the annulus. With the shift of the nucleus from its central position, the vertebrae are no longer held parallel to each other. The fulcrum of the joint, the nucleus, now lies anterior, posterior, or lateral, and the two long arms of the lever, the vertebral surfaces, approximate with widening of the short arms. With widening of the short arms, tension is placed on the intervertebral ligaments with resulting pain. There also occurs simultaneously a shift in the relationship of the facets with tension on their ligaments. Because of the ligament tension pain, and because of the inherent tendency of muscles to protect unstable joints, the muscles become rigid and spastic in an effort to immobilize the involved vertebrae. Thereafter, one of two events occur which result in transient recovery. Either the ligaments stretch to accommodate the newly acquired relationship of the vertebrae, or the nucleus migrates back to its original position near the center of the disc, as a result of release of muscle spasm and the constant massaging action of the vertebrae.

Repeated attacks deepen the rent in the annulus until eventually the nucleus herniates through the rent and lies under the intervertebral ligament, similar to an inner tube protruding through a rupture of the fabric of a tire casing and bulging the overlying rubber. After having established an extensive avenue of escape through the annulus, it is improbable that the nucleus will migrate back to its central position, though it is thought that it might do so. Constant pressure of the extruded nucleus against the intervertebral ligament results eventually in its rupture and the escape of the nucleus from between the vertebral bodies. After the nucleus escapes, the disc has lost the factor intended to carry weight, and the full weight falls on the annulus which, already degenerated, rapidly flattens. The disc also ceases to be a machine, capable of movement in all directions over the ball bearing nucleus, and what movement persists must be a gliding one for which the vertebrae were not constructed. The facets become jammed together as the disc flattens and then develop a degenerative, traumatic type of arthritis.

Whether the symptoms will be confined to back pain or will include sciatic pain will be determined by the location of the rupture of the annulus. If the rupture occurs posteriorly, the nucleus will bulge into the neural canal and, pressing on a nerve root, will cause pain along its course. The nerve root symptoms may subside if the protruding portion of the nucleus liquefies or if it becomes detached and migrates to a quiet area in the neural canal. Repeated protrusions may cause successive attacks. If the rupture occurs laterally or anteriorly, the nucleus escapes into an area where there are no nerve roots, and the symptoms are confined to the back. Late, however, after the disc has flattened sufficiently to narrow the intervertebral foramen, there may be pressure or irritation of the nerve root in the foramen with less well defined radiating pain. In a diagrammatic way the entire syndrome may be visualized in the accompanying chart.

This concept of the pathology of the disc correlates the variant symptoms as described by the patients and explains their mechanical implications. It explains the progression of the symptoms through successive attacks to the final stage of the complaint. It allows one pathological process to explain the divergent courses taken by different patients. It interprets the occasional dramatic relief achieved by manipulative therapy and the excellent results of surgery. It explains the pathology of 75 per cent of the orthopedic species of backache.

Trauma and degeneration are so interwoven in the pathological picture that it is difficult to assign

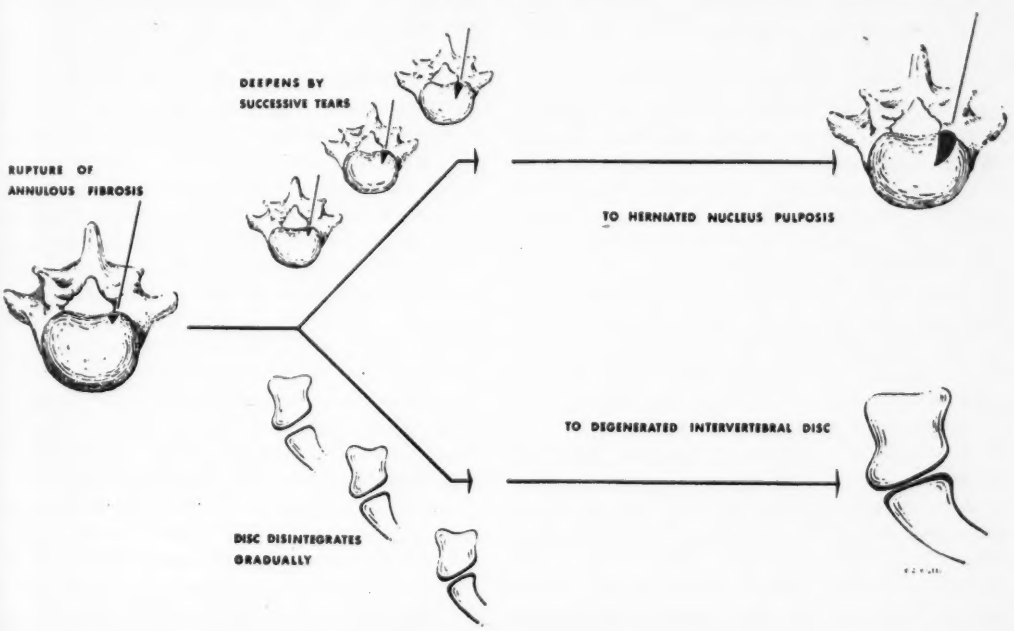
a predominant position to either. Discs in the middle decades of life showing no degeneration are the exception rather than the rule, as Schmorl pointed out, and these are the decades in which most backs become symptomatic. However, pre-existing degeneration is not an absolutely essential factor in the pathology; trauma alone without previous degeneration can be the only factor in the rupture of the annulus. This is proved at operation on the occasional case of herniated nucleus in young adolescents. These adolescent cases are infrequent, and they always give a history of significant strain. One of our patients, a 13-year-old epileptic, on recovering consciousness after a seizure, had severe sciatic pain, proved at operation to be a result of nerve root pressure from a nucleus herniated through a normal annulus. Another patient, a 13-year-old boy, had instant onset of sciatic pain in the course of a heavy lift, and he also had a nucleus herniated through an annulus which showed no pathological changes. Cases at this age, before degenerative changes have been initiated, are rare, but they prove that trauma has an integral part in the pathogenesis.

A homely simile may clarify our thinking about the roles of degeneration and trauma in precipitating symptoms. Termites may have quietly and inconspicuously destroyed the strength of the joists under a floor, and yet for all normal functions the floor remains competent. The presence of this degenerative process is unrecognized and may be evidenced only when a sudden strain, as a fall, results in the floor sinking. If the joists had not been weakened by the termites, the floor would not have given way as a result of the fall. Likewise, if the fall had not occurred, the joists might have remained competent indefinitely. However, the termites probably would have continued their degenerative process of the joists, and the floor would have given way eventually by the application of some minimal force in the course of its normal use.

The degeneration of the annulus in the disc is similar to the weakening of the supporting joists in the floor. The instantaneous rupture of this degenerated annulus, precipitated by a lift, corresponds to the breaking of the joists by a sudden fall. The spontaneous escape of the nucleus without specific trauma is similar to the gradual sinking of the floor due to the slow dissolution of the joists.

The role of trauma in precipitating symptoms becomes an important factor, particularly in compensation cases. Considering the simile of the termite-infested floor and the teaching of Schmorl, one may conclude that in adults there usually is a pre-existing weakness of the annulus and the strain of a lift results in its rupture. Thus both strain and predilection are factors in an attack, as strain and predilection are factors in an inguinal hernia.

The symptoms of this syndrome are as familiar to you as are those of any other well-established pathological process and will not be described. When carefully considered, the symptoms obviously originate from mechanical pathology rather than from a disease process. The symptoms follow the course of mechanical principles, only slightly altered by the



reparative and recuperative faculties of the body, which are not possessed by a machine. Poor recuperative power presages a poor prognosis, once the pathological sequence has been initiated. Repeated attacks, gradually increasing in frequency, severity, and duration, and initiated by decreasingly violent traumata, may be predicted. Varying degree of invalidism are inevitable, not infrequently so marked as to preclude any rigorous occupation or avocation.

In treatment of the initial attacks and early stages, only conservative measures such as rest, support, physical therapy, etc., are indicated. After the course of the syndrome has been confirmed, the choices of treatment are few. However, in view of the benignity of the pathology in relation to life expectancy, the choice must be made by the patient and not be dictated by the physician.

A problem comparable to that of the owner of a worn-out jalopy confronts the patient with a disc syndrome. The car owner has four choices. First, the car may be used until it is functionless, and then nothing more can be expected of it. Second, the capabilities of the car may be established, and somewhat less than its capacity may be expected of it. It may carry a load of 500 pounds at 50 miles per hour without taxing it too greatly; and if a load of only 400 pounds is carried at 40 miles per hour, the car might function for a long time. Third, with a little tinkering such as cleaning the spark plugs, flushing the radiator, adjusting the carburetor, and wiring up the fender and the head lights, the car would probably perform better, at least for a time. Fourth, the car could be taken to a shop for a thorough overhaul and probably could be made proficient for a long time.

Following this simile, the patient who has a disc syndrome may follow similar avenues of approach. First, he may elect to do nothing about his back, continue his normal activities, and accept the occasional bout of pain and disability as the price he must pay for the fun and the work he has accomplished. For a patient of ordinarily sedentary occupation and avocations, this may be the wise solution of his problem. Second, he may establish the functions his back will tolerate and determine to maintain his functional level below that threshold. This may maintain a fairly valuable and comfortable occupational and avocational level for the patient, but it does not prevent the progressive degenerative changes and does imply a degree of invalidism. Third, the patient may accept some tinkering procedures such as a permanent support, exercises, rest periods, a springless bed, and the elimination of excessive strain. These procedures will probably improve his comfort and work capacity, but again they cannot correct or prevent the progress of his pathological problem. Fourth, he may elect the overhaul job which is surgery. The surgical approach should include removal of the disc, if it is protruding posteriorly, and the fusion of the adjacent vertebrae.

X-ray diagnosis is of little aid in making the diagnosis or in determining the advisability of a surgical solution to these patients' symptoms. In fact, dependence on x-ray findings has been much of the reason for mistakes we have made in the past in diagnosis and therapy recommended. In the early and middle stages of this syndrome, there is usually no significant x-ray evidence of pathology, and the diagnosis depends primarily upon history and secondarily on examination. Only late, after the degenerative changes in the disc are far advanced, and usually only after many years of symptoms, does x-ray demonstrate significant narrowing of the disc. Waiting for x-ray evidence of pathology to make a diagnosis or to advise treatment is as unwarranted in these cases as it would be in cases of a torn meniscus in the knee. Given a history of a typical torn meniscus in a knee, no responsible surgeon would fail to make a diagnosis or advise treatment. He certainly could be considered amiss to await x-ray evidence of degeneration in the knee before making up his mind.

The intervertebral disc syndrome is as definite in its symptoms as is the syndrome of internal derangement of the knee, and it should be approached with an equally open mind, both in diagnosis and treatment.

This brings me to the second point I wish to emphasize. If the patient's history of back complaint is typical, it may be accepted almost invariably as prima facie evidence of a disc syndrome. Confirmatory physical findings are reassuring, and x-ray evidence is helpful, but history must be the primary support of the diagnosis. The history must be carefully taken and fully evaluated and, if consistent, confidently accepted.

To substantiate this point, we recently surveyed a comprehensive number of patients whose spines we had fused. Forty per cent of this group were operated upon purely upon the basis of a typical history, without confirmatory physical or x-ray evidence of pathology. Of this 40 per cent, 85 per cent achieved satisfactory results. Sixty per cent were operated upon who had not only confirmatory physical findings but also significant x-ray evidence. Satisfactory results were achieved in 92 per cent of this group. Thus, the results in the group without objective findings to support the diagnosis were essentially as good as in the group with objective findings. This seemed to us to be significant and to prove the point that a typical history of an intervertebral disc syndrome is diagnostic, regardless of physical findings or x-ray evidence.

When we accept the mechanical-degenerative internal derangement of the intervertebral disc as the etiology of the majority of back complaints, and when we accept a typical history as proof of existence of that pathology, we will have come a long way in serving these patients.

Thyroid Activity in Coronary Heart Disease

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There has been little published in the medical literature, to our knowledge, concerning the thyroid status in uncomplicated coronary heart disease, although there is considerable information on the relationship of thyroid status and serum cholesterol[3, 4, 8, 9] and the treatment of coronary heart disease by depressing thyroid activity.[1] This evidence, however, is insufficient to show what the thyroid status is in coronary heart disease.

The reports of Lerman and White,[7] in a limited series of patients under the age of 40 with coronary heart disease, suggested that the thyroid status was in the direction of hypothyroidism. Since this series was small, additional studies in evaluating thyroid status in a larger group were desired by Dr. J. Lerman and Dr. P. D. White. Starr's[15] earlier report of oxygen consumption in eight patients with coronary heart disease was too small for comparative purposes.

The thyroid status may be gauged by means of various tests, none of which in itself is an absolute yardstick. A combination of several of these tests would be of more value as an index to thyroid activity than a single test, since each test measures a different aspect of thyroid activity. This study has employed this principle of a combination of various tests in assessing thyroid function in patients who have had myocardial infarction prior to the age of 40. The tests are as follows:

a. Basal metabolic rate
b. Radioactive iodine excretion
c. Serum cholesterol (total and esters)
d. Serum phospholipids
e. Ratios—cholesterol esters/cholesterol total, and total cholesterol/phospholipids

It is believed that a better insight into the thyroid status is obtained by these several tests, and it is unfortunate that several others were not included; namely, serum protein-bound iodine and thyrotropic hormone in urine (infra vide).

The purpose of this paper is:

a. To present the findings of various tests on thyroid activity on 87 young males who had experienced myocardial infarction.

b. To assess the thyroid status in these individuals.

c. To consider the correlations between thyroid activity as measured by these tests and other measurable variables in the coronary disease group.

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METHODS AND MATERIALS

As a part of a larger and more inclusive study, the thyroid activity was studied by determining basal metabolic rate in 87 males who had experienced a myocardial infarction prior to the age of 40. Of this group, 21 had radioactive iodine studies. The lipid determinations presented in this paper have been discussed elsewhere.[5] These men, without exception, had experienced their infarctions at least six months prior to admission to the Massachusetts General Hospital for study, and all cases except five had returned to some form of activity. In order to study them, each patient was hospitalized for periods varying from 24 to 72 hours. The basal metabolic rate was determined under standard conditions by the thyroid clinic of Massachusetts General Hospital after the patients had been hospitalized the afternoon prior to the determination of the test.

The radioactive iodine excretion studies were done by the technique adopted in the Massachusetts General Hospital, which consists of administering to the patient 100 microcuries of radioactive iodine 131 (half-life 8 days), with 100 micrograms of inert sodium as a carrier. A small aliquot was taken for a standard. Urine was collected for 48 hours, divided in two 6-hour periods, one 12-hour period, and one 24-hour period with a total of 48 hours.

RESULTS

I. Values of Basal Metabolic Rate in Coronary Heart Disease

The results on basal metabolic rate determination on the 87 individuals are summarized in Table I. Unfortunately, matched controls are not available for purposes of comparison.

TABLE I

BASAL METABOLIC RATE IN CORONARY HEART DISEASE

BMR Values	*Number*
−30 to −26	4
−25 to −21	11
−20 to −16	14
−15 to −11	20
−10 to −6	17
−5 to −1	14
+1 to +5	4
+6 to +10	2
+11 to +15	1
Total 87	
Range −30 to +12	
Mean ± st. error −12 ± .9	

The summary illustrates the per cent distribution of various basal metabolic rates in this group which compares closely with the observations reported by Lerman and White.[7] The validity of these findings will be discussed, since the mean basal metabolic rate in the coronary heart disease group is certainly on the low side of accepted normal values. This reduced rate raises the question of whether it is due to: (a) a depressed thyroid activity, (b) failure of end organs to respond to thyroxin or thyrotropic hormone respectively, or (c) a failure of the standardizations used in basal metabolic rate determinations to correct adequately for body build.

II. Radioactive Iodine Studies

The unique avidity of the thyroid gland for iodine provides a valuable means of studying the thyroid by the use of radioactive isotopes of iodine. This principle has been extended to the study of abnormal thyroid physiology (Hertz). The range of radioactive iodine excretion of 21 patients with coronary heart disease is included in Table II, which also presents the radioactive iodine excretion rates of Skanse, et al.[11, 14] in the various manifestations of thyroid activity for comparison with the coronary heart disease group. These results of the radioactive iodine excretion studies do not reveal any change from the euthyroid state.

TABLE II

a. Thyrotoxic	6—32 per cent excretion in 48 hours
b. Myxedematous	74—93 per cent excretion in 48 hours
c. Euthyroid	53—84 per cent excretion in 48 hours
d. Coronary heart disease group	51—78 per cent excretion in 48 hours

III. Serum Lipid Constituents

The values for serum cholesterol levels in hypothyroidism and hyperthyroidism have been the subject of discussion for years, and it is generally accepted that cholesterol rises in hypothyroidism and decreases in hyperthyroidism.[8, 10]

Table III summarizes the lipid constituents in the coronary heart disease group as compared to the normal matched control group in this series, as well as a comparison in hypothyroidism and hyperthyroidism which was obtained from the work of Foldes and Murphy[4] on blood lipids in thyroid disease.

TABLE III

Mg/100 ml. ± ST. ERROR

	No.	*Cholesterol*	*Lipid Phosphorus*	*Cholesterol/Lipid Phosphorus*
Coronary Heart Disease Group	87	286.5 ± 6.6	12.6 ± 0.26	22.8 ± 0.08
Control Group	97	224.4 ± 3.5	11.9 ± 0.13	18.8 ± 0.04
*Hypothyroid Group	7	414.1 ± 29.9	14.3 ± 0.64	28.7 ± 0.57
*Hyperthyroid Group	12	156.2 ± 22.5	7.5 ± 0.61	20.9 ± 1.15

* Obtained from Foldes and Murphy's article on blood lipid distribution in thyroid disease.[4]

There may be some disagreement on the use of the lipids as an index of thyroid function, since it is not known whether the change in lipids is due to the individual's inherent characteristics causing him to be prone to coronary heart disease or to a slight change in thyroid activity. However, these results are included for completeness and will be discussed later in this paper.

IV. Correlations Between Basal Metabolic Rate and Other Variables

The coefficients of correlation were calculated between basal metabolic rate and other variables such as uric acid, 17-ketosteroids, various lipids, age and body build. These correlations are listed in Table IV.

TABLE IV

Coefficient of correlation between basal metabolic rate and	
Uric acid	+.11 ± .11
Cholesterol	+.09 ± .10
Phospholipids	+.10 ± .11
17-Ketosteroids	+.19 ± .11*
Age	−.10 ± .11
Height	−.35 ± .11**
Weight	−.06 ± .11
Endomorphy	−.02 ± .11
Mesomorphy	+.18 ± .10*
Ectomorphy	−.20 ± .11*

* approaching significance
** highly significant (Lindquist, 1942, p. 195)

These correlations indicate: (a) no correlation between basal metabolic rate and uric acid, (b) no correlation between basal metabolic rate and serum lipids, (c) a positive correlation between basal metabolic rate and 17-ketosteroids, and (d) a highly significant correlation in height, and (e) a correlation approaching significance in mesomorphy and ectomorphy. In coronary heart disease each variable is independent of the others, which explains those variables with no correlation.

V. Basal Metabolic Rate and 17-Ketosteroids

The role of 17-ketosteroids in coronary heart disease has been discussed elsewhere by the authors.[6]

The correlation in a positive direction between basal metabolic rate and 17-ketosteroids corroborates the anabolic properties of androgenic materials.

VI. Basal Metabolic Rate and Lipids

Since there is no correlation between basal metabolic rate and the various lipids in this series, this would also tend to show that these individuals are in a euthyroid state. Means,[9] using an example of a series of 70 patients from Mason et al.[8] on thyroid disease, reproduced their graph showing a high degree of association between basal metabolic rate and the cholesterol value in the two opposite pathological states of thyroid activity. Therefore, in euthyroid individuals, non-significant correlations would not be expected to exist, both falling within normal ranges. There are a number of arguments against this thesis by presenting other abnormal conditions such as Addison's disease where blood cholesterol and basal metabolic rate are both low. However, it should be pointed out that in such conditions all body functions are disturbed, and it is not a disease of thyroid, primarily, but a secondary effect.

VII. Basal Metabolic Rate and Physique

In the classification of physique into the three components of endomorphy (softness and roundness), mesomorphy (width, bone, and muscle), and ectomorphy (linearity), the somatotype method of Sheldon[13] was used.

The validity of the finding of the basal metabolic rate on the low side of accepted normal in the coronary group is questioned by the evidence that significant coefficients of correlation were found between basal metabolic rate and other variables of body build (Table IV). It is expected that an adequate correction for a given variable (as is done in basal metabolic rate determinations) would be shown by no correlation between that variable and the basal metabolic rate as computed. It is evident from Table IV that the formula seems to be adequate for the coronary heart disease group for age and weight. However, the significant negative correlation between basal metabolic rate and height indicates that the correction for this variable is inadequate. This assumption is substantiated by the negative correlation between basal metabolic rate and ectomorphs and the positive correlation between basal metabolic rate and mesomorphy.

DISCUSSION

The evidence presented here indicates a euthyroid status in the coronary group. As has been pointed out, the mean basal metabolic rate in the coronary disease group is on the low side of accepted normal values. However, the coefficients of correlation indicate that the formula for basal metabolic rate has not been corrected adequately for this group. The basal metabolic rate is the percentage variation of oxygen consumption of an individual above or below the expected (calculated) oxygen consumption under basal conditions.[2] The expected oxygen consumption, or standards, have been empirically determined by considering the variables, age, sex, and surface area; the latter as derived by the height and weight of the individual, of a limited number of individuals. Of these variables, surface area would be the most prone to error, especially in the shortest and tallest individuals (the extremes of the distribution). In these individuals ofttimes the weight is out of proportion to stature and would, therefore, increase the error in estimating surface area. The coronary disease group has been shown to be predominantly mesomorphic; their linear measurements are decreased while the lateral measurements are increased. Seltzer[12] has shown that oxygen consumption in linear individuals is greater than their "lateral" counterparts; this observation is substantiated in this study by the findings of a negative correlation between basal metabolic rate and ectomorphy and the positive correlation between basal metabolic rate and mesomorphy. Therefore, it is reasonable to suggest that a basal metabolic rate of –12 in the predominantly mesomorphic individuals, as found in the coronary heart disease group, is normal for this type of individual and is in keeping with the theoretical considerations.

Probably the best work on the interrelationship of the lipids in thyroid disease is that of Foldes and Murphy.[4] In general, these authors also confirmed the findings that serum cholesterol is inversely proportional to thyroid activity, and they extended it to include other lipids. Thus, not only is cholesterol found to be significantly increased in hypothyroidism, but also cholesterol esters and phospholipids. The cholesterol esters/total cholesterol and total cholesterol/phospholipid ratios were significantly increased.

In hyperthyroidism these changes were not constant or significant (Table IV). The plasma phospholipids were decreased significantly, and the cholesterol was decreased absolutely. The resulting total cholesterol/phospholipid ratio was not different from the normal values given. The serum cholesterol (total), serum phospholipids and the total cholesterol/phospholipids ratios were increased in both the hypothyroid state and the coronary disease state. This would suggest moderate decrease in thyroid activity in the latter state. However, on analysis, the essential difference is the degree of increase of both constituents.

In hypothyroidism the serum cholesterol rises to unusually high levels, while the serum phospholipids do not rise proportionally; this accounts for the high total cholesterol/phospholipid ratio. In coronary heart

disease, the serum cholesterol is moderately increased and the phospholipids are but slightly increased, accounting for the moderately elevated cholesterol/phospholipid ratio. These various lipid components are significantly higher in the hypothyroid state, and these results would therefore reinforce the evidence submitted that there is no significant change from the euthyroid state in coronary heart disease.

In the small series (21 individuals) on whom radioactive iodine excretion studies were done, the results did not reveal any change from the euthyroid state.

SUMMARY AND CONCLUSIONS

1. The thyroid status in coronary heart disease was assessed through the use of multiple tests: (a) basal metabolic rate, (b) radioactive iodine, (c) interrelation of various serum lipids.

2. The basal metabolic rate was, on the average, on the low side of accepted normal. However, it was shown by correlation coefficients that such a reading is normal if morphologic characteristics are accurately considered and corrected when surface area is assessed.

3. From the studies made above, it was concluded that there is no alteration of thyroid activity from the euthyroid state in males who have experienced coronary heart disease prior to the age of 40 and whose present average age is 38.5 years.

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WHATEVER America hopes to bring to pass in the world must first come to pass in the heart of America. The peace we seek, then, is nothing less than the practice and fulfillment of our whole faith among ourselves and in our dealings with others. This signifies more than the stilling of guns, easing the sorrow of war. More than escape from death, it is a way of life. More than a haven for the weary, it is a hope for the brave. This is the hope that beckons us onward in this century of trial. This is the work that awaits us all, to be done with bravery, with charity and with prayer to Almighty God.

—Dwight D. Eisenhower

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

FREEDOM VERSUS SOCIALIZATION

In common with most other institutions of higher learning, the nation's medical schools face a problem. They must have more money if they are to do their tremendously important job. This fact was recently recognized by President Eisenhower, when he opened a 10-million-dollar campaign within industry and the medical profession which is designed to help the schools overcome deficits threatening their programs.

For many years we have heard an incessant demand by certain segments of society that the government should provide the necessary money and subsidize the schools. The President demolished that idea in a letter to the head of the National Fund for Medical Education in which he said that the medical school problem "should be solved through private rather than governmental means. Excessive reliance on government violates the essential principle of our free enterprise system. It falls, then, upon American business to assume a greater share of the responsibility for maintaining the institutions essential to our national health."

This reversal of the course pursued by the two previous administrations should receive the acclaim of all who still believe that the private enterprise system is the best for the American people. Certainly it should be obvious to all that government financing of medical education (and any other type of education) would involve government direction of how and where the money was spent. Advocates of government subsidization of education seem willing to risk bureaucratic dictation in return for handouts from the national treasury. They do not see, or perhaps don't care, that the whole medical educational picture could be catapulted into socialized medicine. And categorically, the whole educational system might be socialized.

Nothing like that is going to happen under the Eisenhower administration. The proponents of socialization may weep and wail and gnash their teeth, but the vote of the people last November gave a clear mandate: "Turn, Boys, We're Going Back"—to free enterprise.—*Topeka Daily Capital, June 4, 1953.*

HEALTH AND SOCIALISM

Mrs. Oveta Culp Hobby, secretary of the Department of Health, Education and Welfare, revealed that only 10 per cent of the amount needed by the voluntary National Fund for Medical Education is now available.

The fund was established to help medical schools pay their bills and to train more doctors and other health personnel. Mrs. Hobby told the American Medical Association that the schools' "financial crisis is still growing."

The fact that educational facilities for our future doctors cannot be given more than tiny bits of aid through voluntary contributions by corporations and individuals is to be regretted.

More to be regretted, however, is the fact that any move to allow the government to help the schools is immediately termed "Socialistic" by the organized medical profession and Mrs. Hobby herself.—*Kansas City Labor Bulletin, June 26, 1953.*

HUMAN TOUCH IS ESSENTIAL

The newly-elected president of the American Medical Association has said, "although medical science has produced many drugs of near-miracle effectiveness in the last decade, I find that among the most potent of all medicines is still the human touch."

Here is one of the strongest of all the many arguments against socialized medicine or its variant, government dominated and directed medicine. Wherever these forms of medicine have been tried—and England is the best example for us Americans—the human touch has been largely destroyed. Neither doctor nor patient is any longer a free agent. The doctor must practice "by the book"—and the book is written and its rulings enforced by bureaucrats and politicians. The patient must also follow the book, and take whatever kind and quality of medical care the politicians in power decide he is to have, or do without. Doctors are overworked, under some sort of a panel system, and the result is assembly line medicine. There is little time for providing that all-important human touch—even as there is little time for research and study. The loss in both human values and scientific values is beyond measurement.

The goal of American medicine is to keep the human touch, to continue to give us the best medical care on the face of the earth and to gradually solve the economic problems of illness. That is what is being done here under the free system.—*Topeka Daily Capital, July 18, 1953, reprinted from Industrial News Review.*

PRESIDENT'S PAGE

DEAR DOCTOR:

Progress in medical education is the basic premise upon which all medical societies are founded, and it has been through the constant expansion and widening of the base of that premise that our Society has survived and flourished.

With the ever increasing cost of producing sound medical teaching and research, individually and collectively, we are going to be faced with a greater responsibility in every aspect of medical education.

A few years ago, I happened to be present when the president of the A.M.A. called the House of Delegates into session and announced what they considered to be the most important decision ever made by the Board of Trustees, and that was a donation of some $500,000 to the American Medical Educational Foundation. At that time they encouraged individual donations to this fund, either by sending contributions directly to the American Medical Educational Foundation or to the medical school of their choice. Those who choose to send their contributions to the A.M.E.F. can designate the medical school to which their grants are made.

In 1952 a total of 155 doctors of Kansas made contributions, either directly or indirectly, to the medical school of their choice. Of the 155 contributions, only 53 were made to the University of Kansas School of Medicine. The total sum of these contributions was $5,226. In comparison with contributions to other medical schools, both in number and in size of contributions, our university did not fare very well.

As an expression of pride in our state and in our university medical school, it is my hope that in the report for 1953 each and every member of the Kansas Medical Society will be listed as financially supporting the American Medical Educational Foundation, and that all or part of each contribution will be earmarked for the University of Kansas School of Medicine.

EDITORIAL COMMENT

The Doctor Draft Act

The 83rd Congress amended the Universal Military Training and Service Act in sections pertaining to physicians and other specialized services in what is known as Public Law 84. It was signed by President Eisenhower and is currently in effect and will operate for a period of two years, beginning with July 1, 1953, and concluding on June 30, 1955. The new law, while retaining the general structure of the previous act, revises numerous sections, many of which will serve to benefit the individual doctor as well as communities.

Along with items that were not altered from the previous law is that it will still be necessary for all physicians, dentists, and veterinary surgeons who are not members of an armed service reserve component, and who are under 50 years of age, to register with their local draft boards. Men graduating from medical school will have 10 days in which to register, and may then ask for deferments for one year in which to complete their internships. All physicians must register under the doctor draft, even though they have previously registered for the regular draft. However, those who have registered under the doctor draft previously need not repeat that process.

Service liability is retained to the 51st birthday, as before. Unchanged also is the $100 monthly extra pay for physicians, which will continue for those who are in the service during the next two years. Unchanged also are deferment mechanisms. As before, deferments are temporary and may generally be based only upon community hardship. Because of the relatively high rank and the bonus payment received by the medical officer, it is quite rare that he may obtain a deferment on the basis of personal hardship. Relatively few deferments will be granted residents. The practicing physician, if deferred at all, may expect this to be based on civilian needs for his services and, as such, it is for a short period of time to enable the community to obtain a replacement. As before, deferment requests are handled through the Kansas Volunteer Advisory Committee, of which Byron J. Ashley, M.D., Topeka, is chairman.

There are many small differences between the previous and the new regulations, but the primary alterations in the law occurred in two areas. Priorities have been changed. Previously, the doctor who had received a portion of his education under either A.S.T.P. or V-12 was permitted to count only such service as occurred following his government schooling. That has now been changed to where all service since September 16, 1940, shall be included in tabulating previous service, regardless of whether this came before or after government schooling. An illustration of this change might be drawn from the hypothetical case of the young man who served two years as a pilot in the Air Force and then went to medical school under A.S.T.P. The old act gave him no credit for previous military service and placed him in Priority I. Today such a physician would be in Priority IV.

Priority I remains as it was except for the explanation above. This includes those doctors who had schooling under A.S.T.P. or V-12 or those doctors who were deferred from service to continue their medical education and who have served less than 90 days, including internship or other periods of education since September 16, 1940.

Priority II has been changed. The former law classified this priority as those physicians who received government education under A.S.T.P. or V-12 or who were deferred from service to complete their education and who had served at least 90 days but less than 21 months. This has now been altered in the service requirement section to include those doctors who have served 90 days to 17 months. The difference here is in the four-months period. Today the doctor formerly in Priority II with more than 17 months of service will be classified in Group IV.

Priority III in unchanged. This includes all physicians under the age of 51 years who are not members of a reserve component and who have not served with the Army, Navy, Air Force, Marine Corps, Coast Guard, or United States Public Health Service since September 16, 1940.

Priority IV contains all other physicians under the age of 51 years except those in the reserve components. It consists of the veterans of World War II. Those in Group IV will be particularly interested in a further important change to the effect that no physician who has served 21 months may be recalled during the life of this act except in the case of a national emergency. It is possible to recall certain Group IV physicians, but since the lower categories must be nationally exhausted before this may be reached, it appears doubtful that any Group IV physicians will be called.

The second major change concerns the length of service that may be required. In most instances, the new tabulation favors the physician in that it shortens the period of service. According to the new law, the doctor who has credit for less than nine months, if called, will be required to serve 24 additional months. If his previous service was nine to 12 months, he will be recalled for 21 months. The

doctor who has previously been with the armed forces 12 to 15 months can be recalled for just 18 additional months, and, if he had 15 to 21 months previous service, he may be recalled for 15 months only. Then, as stated before, anyone having served more than 21 months is not liable for recall.

Of even more interest is that this is retroactive for those now in the service. The officer who would benefit by these new provisions may file an application through his branch of the service, and, depending upon his individual situation, will have his period of service reduced or terminated. Should he have completed his assignment, his release will be arranged as rapidly as possible, but in no event later than 90 days. It should also be noted that upon the completion of his tour of duty, the medical officer will, in all instances, be permitted to resign his commission and, in most situations, will be required to do so.

To complete the story of the new draft law, a few other miscellaneous items should be mentioned. This act makes it possible to count as prior active duty wartime military service spent with any World War II ally of the United States or in nonmilitary duty prescribed for conscientious objectors or service spent with the Panama Canal Health Department during World War II. It also is possible under this act to commission alien physicians as medical officers.

Finally, doctors shall be commissioned in grades commensurate with their professional education, experience, or ability. However, since the doctor draft law is part of the Selective Service Act, doctors are subject to the Selective Service system up to the time they accept a commission. Thereafter, their course is administered under regulations laid down by the armed forces. Selective Service is the means for obtaining induction, but once this has been satisfied the various branches of the service determine the grades that will be granted.

Legal Aspects of Sterilization

This is the second in a series of editorials that shall continue in subsequent issues on legal aspects of the practice of medicine. The first of this series, on the subject of fee splitting, appeared last month.

Physicians are frequently confronted with a request to perform a sterilization procedure. This article is prepared to advise the profession concerning the legality of such operations in this state. Kansas law, similar to the law on this subject in other states, is inconclusive. Therefore, even after the law is reviewed, the physician's liability remains in doubt. The rather long preliminary discussion may, at first, appear to be beside the point in question. It seems necessary, however, to include this since conclusions are drawn from the following facts.

Kansas law covers the subject of sterilization under Chapter 76 of the General Statutes of 1949, beginning with Paragraph 149 and continuing through Paragraph 155. This law appears to have been on the books since 1913. It is important to note that the chapter heading is State Institutions. The above mentioned sections describe the manner in which sterilization operations may be performed upon inmates of institutions.

The superintendent or warden of certain named institutions in Kansas, if he has a person under his custody who he believes would be physically or mentally benefited thereby, or if he believes that the ". . . procreation by such inmate would be likely to result in defective or feeble-minded children with criminal tendencies, and that the condition of such inmate is not likely to improve so as to make procreation by such person desirable or beneficial to the state . . ." may request such person to be sterilized. This request is made in writing, and a Board of Examiners, consisting of the chief medical officer of such institution, its governing board, and the secretary of the State Board of Health, shall hold a hearing. The patient and his guardian are entitled to present testimony and shall be permitted legal advice in their defense. If, after such hearing, the board rules that sterilization shall be performed, a surgeon shall be selected to perform the necessary operation, and payment shall be made out of the budget of the institution involved.

If the operation is performed upon a male, it may be either a vasectomy or an asexualization. If a female, it may be either a salpingectomy or an oophorectomy. Then the final section dealing with this subject, 76-155, states the following: "Except as authorized by this act, every person who shall perform, encourage, assist in or otherwise promote the performance of either of the operations described in this act, for the purpose of destroying the power to procreate the human species, unless the same shall be a medical necessity, shall be fined not less than $100 nor more than five hundred ($500) dollars, and imprisoned in the county jail not less than six months nor exceeding one year."

The above discussion appears to be necessary because two distinct legal opinions prevail on this subject. One thought is that this law applies only to inmates of state institutions, and the last quoted section implies that no inmate of a state institution may be sterilized except in accordance with the above procedure. It is therefore reasoned that persons who are not inmates of state institutions are not covered by the provisions of this act, and, since sterilization is nowhere else mentioned in the Kansas law, there

are no legal provisions either permitting or denying the surgeon the right to perform such operations.

There are attorneys who hold a different opinion. They reason that the law specifically states that sterilization may be performed upon inmates of state institutions and that, by reason of the state having carefully outlined the procedure through which this may legally be performed, it must then be presumed that unless the operation conforms to all sections of this statute it cannot otherwise be done legally in this state. In other words, Section 155 says, "Except as authorized by this act . . ." and that all other sterilization procedures are illegal.

Regardless of which legal view the physician prefers to accept, one factor is certain. If the patient's health or the saving of his life requires such surgical procedures to be performed, and if the purpose of performing such operation is not for the prevention of procreation, then there is no doubt as to the legality of the procedure. The doubt arises over sterilizations that are performed for reasons other than medical necessities.

There is at least one legal opinion of interest on this subject in which the patient sued on a theory of breach of warranty because the operation did not have the desired effect. The principal issue was not concluded since it was thought that the operation was necessary to save the life of the plaintiff's wife.

In the absence of court rulings, the Kansas situation remains in doubt. It is a subject about which the individual opinions of the court might vary. It is also a subject upon which there is no established fact. Therefore, it appears advisable to suggest that, even if written consent of the husband and wife is obtained, that fact is no protection should the operation be declared illegal. In other words, the physician who performs a sterilization procedure upon the request of his patient, and who cannot establish that this was performed on the basis of a medical necessity, has no legal protection in the signed release. Should the operation be ruled illegal, the fact that it was approved by the patient is no protection.

Finally, then, it appears that the Kansas courts might rule conservatively, and the physician who sterilizes an individual for any but definitely therapeutic reasons runs the risk that he is performing an illegal operation. This fact is not changed because the patient consents in writing to the operation or even requests it.

American Medical Educational Foundation

Among the more interesting recent projects of the American Medical Association is the organization known as the American Medical Educational Foundation. Every physician is familiar with the purpose of this foundation, which is to aid medical schools through voluntary contributions. The money is distributed to each of the 70-odd approved medical schools in this country on the basis of two separate formulas. And, furthermore, each contributor may assign his gift to the school of his choice if he cares to do so.

The need for establishing the American Medical Educational Foundation stems from the financial distress experienced by many medical schools. This,

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

James E. Crockett, M.D.
1919 Olathe Boulevard
Kansas City, Kansas

Albert F. Crumley, M.D.
619 North Main
Ottawa, Kansas

Donald R. Germann, M.D.
4603 Melvin Street
Kansas City, Kansas

William T. Holland, M.D.
Winter VA Hospital
Topeka, Kansas

Warren L. Krump, M.D.
312 Northeast Avenue
Oberlin, Kansas

Lester E. McGonigle, M.D.
Potwin, Kansas

John W. McKay, M.D.
3916 Willow Drive
Wichita, Kansas

Charles W. Merten, M.D.
630 Santa Fe
Augusta, Kansas

William S. Mowrey, M.D.
Luray, Kansas

William L. Padgett, M.D.
301 South Maple
McPherson, Kansas

Richard V. Polson, M.D.
722 South Grove
Wichita, Kansas

Marion A. Throckmorton, M.D.
1026 Perry
Wichita, Kansas

Richard N. Todd, M.D.
916 West Pine Avenue
El Dorado, Kansas

coupled with the long-standing hysteria over the shortage of physicians, has created considerable political interest in federal subsidies. Organized medicine, for reasons that are quickly obvious to everyone, opposed the general theory of federal grants, and, to obviate the oft-repeated criticism that medicine opposes everything but offers no concrete solutions, the A.M.A. undertook this project. Two full years have elapsed since its inception, and a recent tabulation of the work of this foundation appears to be of considerable interest and is summarized below.

Organizations such as pharmaceutical houses and other corporations have contributed handsomely, but included in this report are only those contributions received from practicing physicians. In the year 1951, the doctors of the United States gave $91,000, but last year had increased their contributions to a total of $291,000, representing a growth of 210 per cent. The list of individual contributors shows a still greater gain. In 1951, 1,876 separate checks were received. The list of individual physician donors in 1952 had grown to 7,259. The report listed the number of donations according to the several states. In many, including Kansas, the number of donors is very small, but there are examples in which a large percentage of all physicians made contributions. In Indiana, for instance, 1,217 individual doctors contributed to this fund last year. Nearer home, Colorado may be cited with 400 contributions totaling $13,611.50. Nebraska is even more surprising with 474 contributions totaling $49,146.25.

Against the above, Kansas has not been impressive. In 1952, a total of 19 doctors made gifts to medical education through this fund which totaled $840. While this is certainly not indicative of the interest Kansas physicians have in medical education, nor in the preservation of freedom in medical schools, it represents a lack of interest in a most worthwhile project that should be supported by every Kansas doctor. The JOURNAL is advised that contributions may be of any size the physician wishes. The amount of money is not nearly so important as the number of donors, and in an effort to improve the Kansas situation in this regard it is respectfully requested that each doctor plan to take a part in this program during the coming year.

Even though the above does not place Kansas in an attractive position, there are at least three things that should be said to complete the story. The first, being in the form of an alibi, is that many states have records equally as poor. Of more importance, however, is the fact that the above does not present the total picture. Not listed among contributions credited to Kansas doctors were 136 gifts made directly to aid medical schools outside this fund. Even if this figure is combined with the 19 listed above, it is still not good.

The report also tabulates how much money was donated directly to each of the medical schools. Here, again, the Kansas picture is not what it should be because through direct contributions the University of Kansas School of Medicine received $5,226 from 53 contributors located throughout the United States.

There is one bright side to this picture in that numerous county medical auxiliaries have contributed sizable sums to the American Medical Educational Foundation. If this subject is of sufficient importance to the Auxiliary, with its relatively limited funds, it certainly should be wholeheartedly supported by the profession. The JOURNAL wishes to commend this project to the doctors of Kansas. If anyone desires further information on the subject, it may be obtained through writing the Executive Office of the Kansas Medical Society.

PROGRAM FOR IMPROVEMENT

The following nine points were listed by the new president of the American Medical Association, Dr. Edward J. McCormick, Toledo, as an outline for his program of continued improvement in medical care for the nation:

1. The distribution of doctors is a problem. Much has been done by medical organizations to solve it. Placement services are now in existence in 37 states. Of these, 32 are operated by medical societies. It is important to the future of medicine that every community have access to a physician. Medicine must actively aid those communities which are trying to attract doctors.

2. Over 600 of our county medical societies now have 24-hour emergency call services. I urge all others to support such a system.

3. Every medical society must have a strong and fearless mediation committee to hear patients' complaints. These must not be whitewash committees. They must be true to the purpose of their founding by reprimanding and disciplining physicians found guilty of exploiting their patients. Only in this way can public confidence in medicine be maintained.

4. Physician and hospital relationships must be clarified and steps taken toward mutual cooperation. I advise the formation of physician-hospital committees by state and county medical societies to work toward better relations in local communities. This has already been done with some success in some states.

5. Every county society should become an active unit in the nationwide effort to develop and expand voluntary health insurance. We must find ways of

providing protection against catastrophic illness and coverage of older age groups.

6. Too many physicians have been isolationists within their communities. Local societies should encourage each individual member to participate in some civic undertaking. We physicians should be rendering health leadership in all service clubs, fraternal organizations, parent-teacher groups, church associations and unions.

7. Every doctor must be brought to realize that good public relations begins in his or her office—that the way in which they treat patients reflects for good or ill on the entire profession. Medical societies are frequently hampered in their efforts to build public understanding by the doctor who overcharges, the doctor who rudely refuses to answer a night call no matter how urgent, or the doctor who keeps patients waiting for hours in his reception room without any explanation.

8. There are some newspaper and radio people who honestly believe some of the untruthful charges which have been made against medicine. All county and state societies should make continued efforts to develop a close association with writers for press, radio and television.

9. There is a need for unity within the profession. I have noticed a distressing regression toward petty internal wrangling, charges and countercharges, and divisive activities by various groups within the profession.

COUNTY SOCIETIES

Members of the Riley County Medical Society were hosts to the Golden Belt Medical Society at a meeting held at Manhattan on July 9. For the afternoon program Dr. Z. Miles Nason, Kansas City, spoke on "Medical Treatment of Alcoholism," and Dr. Maurice Snyder, Salina, discussed "Medical Management of Hypertension." A dinner and business session followed the scientific program.

Members of the Brown County Medical Society entertained their wives and medical assistants at a dinner meeting at the county hospital, Hiawatha, on July 3. Mr. Rueben M. Dalbec, executive assistant of the Kansas Medical Society, Topeka, was speaker of the evening, using public relations as the subject of his talk. A business session and a tour of the newly completed wing of the hospital concluded the meeting.

Dr. Roscoe T. Nichols, Hiawatha, who has completed 51 years in medical practice, was guest of honor at a dinner given at the Hotel Redmon, Hiawatha, on June 29, by the Brown County Medical Society. Fifty physicians and their wives from northeast Kansas were present. Dr. Winstan L. Anderson, Atchison, councilor for that district, was toastmaster. Tributes to Dr. Nichols were given by Dr. George Thacher, Waterville; Dr. Clemens Rucker, Sabetha; Dr. A. E. Cordonier, Troy, and Dr. Fred Wrightman, Sabetha. A response was given by Dr. Nichols.

Chest Physicians Organize

A Kansas Chapter of the American College of Chest Physicians was recently organized, and the following officers were elected to serve for the first year: president, Dr. Charles Pokorny, Halstead; vice president, Dr. A. L. Ashmore, Wichita; secretary-treasurer, Dr. C. J. W. Wilen, Manhattan, and program chairman, Dr. Andre Baude, Topeka. Governor for the state of Kansas is Dr. C. F. Taylor, Norton. At present there are 20 members residing in Kansas.

The College of Chest Physicians, founded in 1935, is an international organization. It endeavors to promote interest and research in diseases of the chest and the allied fields of cardiology, bronchoscopy, and radiology. The official journal of the organization is *Diseases of the Chest,* published monthly. It is the intention of the Kansas unit to provide a program on some phase of chest disease at the annual meetings of the Kansas Medical Society.

Inquiries regarding membership should be addressed to Dr. C. F. Taylor, State Sanatorium, Norton, Kansas.

To Organize Pediatric Society

A meeting for pediatricians in Kansas, and all physicians interested in pediatrics, will be held at Emporia on September 19, and plans for the organization of a Kansas Pediatric Society will be discussed. Arrangements are being completed by a committee composed of Dr. C. T. Hinshaw, Wichita; Dr. William Crouch, Topeka, and Dr. David R. Davis, Emporia.

A scientific program, presented by out-of-state pediatricians, will begin at 10:00 o'clock and continue through the day, with a two-hour luncheon period devoted to round table discussions. The evening program will include a social hour, banquet, and business session.

Physicians interested in attending are asked to write immediately to Dr. David R. Davis, 103 Gazette Building, Emporia.

ACTIVITIES OF MEMBERS

Dr. William C. McDermott, who has been practicing in Hays since November, 1952, has moved to Phillipsburg and is specializing in surgery there.

Dr. E. Grey Dimond and Dr. T. K. Lin, of the University of Kansas Medical Center, are co-authors of a paper, "Reliability of the Grading of Cardiac Murmurs," which was published in the July issue of *GP*, the publication of the American Academy of General Practice.

The Topeka Medical Center announces that Dr. John E. Sweeney is now a member of its staff, specializing in internal medicine. Dr. Sweeney had been practicing in Fargo, North Dakota.

Dr. John F. Coyle, Coffeyville, who is now serving at Gunter Air Force Base, was among volunteers who administered gamma globulin to 30,000 children in Montgomery, Alabama, to combat an outbreak of poliomyelitis.

Dr. Mary T. D. Glassen, Phillipsburg, announces that Dr. Barbara Calderwood is now associated with her in practice. Dr. Calderwood, who was graduated from the University of Kansas School of Medicine in 1952, recently completed her internship at Menorah Hospital, Kansas City, Missouri.

Dr. James E. Henshall, Osborne, has been appointed coroner for Osborne County to succeed Dr. W. K. Nickell, who was recently called into military service.

Dr. G. M. Martin has returned to his position as director of the Division of Maternal and Child Health, Kansas State Board of Health, after an absence of a year spent at Harvard University acquiring a master's degree in public health.

The city of Ellsworth observed "Dr. Alfred O'Donnell Day" in June in connection with the dedication of the new addition to the Ellsworth County Veterans' Memorial Hospital. Dr. O'Donnell, who retired in 1948 after practicing in Ellsworth for 41 years, was honored for his participation in many civic affairs.

Dr. William C. Menninger, Topeka, was one of 17 specialists throughout the nation selected to serve as senior civilian consultant to the Surgeon General, Department of the Army, effective July 1, 1953. Dr. Menninger will be consultant in psychiatry and neurology.

Dr. Farris D. Evans, Wichita, was re-elected department surgeon of the state V.F.W. organization at a recent meeting held in Goodland, and he was also elected delegate to the national encampment to be held in Milwaukee this month.

Dr. Henry G. Hurtig, Hanover, who retired several years ago because of poor health, was the subject of a feature story in the *Hanover News* on July 3 in observance of his 69th birthday. Before his retirement, he had practiced in Hanover more than 30 years.

Dr. Fred W. O'Donnell, Junction City, was one of the speakers at the recent centennial celebration at Fort Riley.

Dr. William E. Brownlee, who has been practicing in association with his father, Dr. John J. Brownlee, in Hutchinson during the past four years, has begun a four-year residency in surgery at the University of Kansas Medical Center.

Dr. Laurel G. Case, who was recently called into service, announces that his office in Enterprise will be kept open during his absence by Dr. T. L. Lothman. Dr. Lothman is a graduate of the University of Kansas School of Medicine and recently completed his internship there.

Dr. Mary T. D. Glassen, Phillipsburg, spoke on the subject of health before a recent meeting of the county farm bureau at the Logan high school auditorium.

Dr. Ben L. Myers, who retired from active practice in Iola after suffering a stroke last fall, is visiting in San Francisco and is planning to make his home in that vicinity.

Dr. Otis H. True, who has been practicing in Hays, has moved to Kansas City to take special work at the veterans' hospitals there and in Leavenworth.

Dr. R. Burnley White, who has been practicing in Hanover, has announced that he will open an office in Phillipsburg late this month.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was one of the delegates from the United States to attend the World Health Assembly in Geneva, Switzerland, this summer.

Dr. Arnold M. Pederson, who recently completed a year at the State Sanatorium, Norton, in special training in diseases of the chest, opened an office last month in Plainville in association with Dr. Vale Page. Both were 1951 graduates of the University of Kansas School of Medicine.

Dr. F. S. Brenneman, who has been practicing in Moundridge, is on his way to Pago Pago, Samoan Islands, for a two-year term in public health work.

Dr. R. R. Nevitt, who has been practicing in Moran for 12 years, has moved to Fort Scott and is practicing there at the Fort Scott Clinic.

Dr. Florence Friesen, who has practiced in Greensburg for seven years, after spending 25 years as a medical missionary in India, has moved to Moundridge and has taken over the office formerly used by Dr. F. S. Brenneman.

Dr. Alfred M. Tocker, formerly of the department of surgery at the University of Kansas Medical Center, has opened an office for private practice in Wichita.

The Eddy Clinic, Hays, announces that Dr. Eugene Siler, formerly of Topeka, is now a member of its staff.

The office of the late Dr. Harold J. Bagby, Coffeyville, has been reopened by his son-in-law, Dr. Robert G. Wood, a graduate of the University of Kansas School of Medicine who recently completed his internship at Providence Hospital, Kansas City.

Dr. George L. Basham, who has been practicing in Eureka since his return from service in the Navy during World War II, has moved to Abilene and is now practicing there.

Dr. Joseph E. Gootee, who has been serving in the Air Force, has been released from the service and has returned to the practice of anesthesiology in Topeka, in association with Dr. Floyd C. Taggart and Dr. W. O. Martin.

With progressive improvement in treatment, fewer tuberculosis patients are dying of the disease. Therefore, for isolation treatment in hospitals, for aftercare, and for rehabilitation, we will probably continue to require more, not fewer, beds for some time to come.—*Public Health Reports, July, 1952.*

DEATH NOTICES

Charles Mooney Fitzpatrick, M.D.

Dr. C. M. Fitzpatrick, 74, an active member of the Saline County Medical Society, died in Salina, July 10, after several months of illness. He specialized in radiology and had practiced in Salina since establishment of a department of radiology at St. John's Hospital in 1915. He was a graduate of the University Medical College of Kansas City. During World War I, Dr. Fitzpatrick served in the medical corps of the Army. He was a member of the Radiological Society of North America, Inc.

Clyde O. Merideth, Jr., M.D.

Dr. Clyde O. Merideth, 46, president of the Lyon County Medical Society, died at his home in Emporia, July 26, after an illness of several months. He was graduated from the University of Kansas School of Medicine in 1931, and for five years practiced in Elkhart before opening his office in Emporia. He specialized in obstetrics and gynecology.

Dr. Merideth was greatly interested in the Boy Scout organization and was president of the Jawhawk Council. He had received virtually every award given to Boy Scout leaders.

Tumor Conference*

Problems in Diagnosis and Treatment of Bone Tumors

Edited by H. I. Firminger, M.D., and Irwin Joffe, M.D.**

Dr. Helwig: This conference should serve to illustrate the difficulties in the diagnosis of certain lesions of bone clinically, roentgenologically, and pathologically. May we hear about the first case?

CASE NO. 52-112

Dr. L. O. Litton: The first case is that of a 15-year-old white girl, admitted to the hospital on October 26, 1952, complaining of pain and swelling over the lateral posterior aspect of the right fibula of about 2½ months' duration. Since the onset of this complaint, both swelling and pain had increased. Past history revealed "growing pains" in the right thigh and calf about one year prior to admission.

The physical examination was essentially negative except for examination of the right lower extremity. There was an indurated 4 by 8 cm. mass over the posterior lateral aspect of the head of the right fibula. There was some redness around this area, and it was very tender. There was no lymphadenopathy. The chest was clear to percussion and auscultation.

The results of urinalysis were normal. Blood count revealed 4,260,000 red cells, 8,350 white cells, 73 per cent hemoglobin, and normal differential. X-ray examination showed a rather characteristic destructive lesion, which was interpreted as osteogenic sarcoma. The chest x-ray was negative.

On the day following admission the mass was biopsied, and this revealed osteogenic sarcoma. On November 3, 1952, a mid-thigh amputation was done, and the patient's postoperative condition is very satisfactory.

Dr. Helwig: Dr. Tice, would you please discuss the roentgenograms in this case?

Dr. Tice: Several points are worth stressing. First, there is definite bone destruction at the upper end of the fibula. Second, there is bone formation out in the soft tissues arising from the upper end of the fibula, adjacent to the destroyed area. Another point is that the tumor is located about the knee; most osteogenic sarcomas occur near the knee, in the lower femur, or upper end of the fibula or tibia. The last point to be emphasized is that the tumor has occurred in a young person. Because of the characteristic x-ray picture, we diagnosed it as an osteogenic sarcoma. There is one thing lacking that does tend to make a lesion of this type characteristic, and that's the formation of a triangle at the lower end of the tumor, the so-called Codman's triangle, which is not invariably present.

Dr. Helwig: Would you like to comment on the question of preservation of the shadow of the old shaft?

Dr. Tice: The old shaft is often preserved; as a matter of fact, I can't see any break in the shaft. The break that I see in the films seems to be a central type of destruction. Another point that is sometimes stressed is the manner of laying down of new bone. We look for the sunburst effect, spicules of newly-formed bone oriented at right angles to the shaft; a hint of that effect is seen in these films, but this finding is not required in order to make the diagnosis. Kolodny,[1] who summarized a group of these cases, found it in 18 per cent of his cases; thus, any bone formation, whether it is the sunburst pattern or simply laid down in an amorphous manner, is of significance. And the most important finding of all is not the bone formation, but bone destruction; many of these tumors are purely osteolytic.

Dr. Helwig: Thank you, Dr. Tice. Dr. Boley, would you tell us about the pathological findings in this case?

Dr. Boley: Examination of the gross specimen revealed a friable, dull gray, fungating mass 5 by 3 cm., affixed to the head of the fibula. The bulk of the tumor lay outside the cortex of the bone, elevating the periosteum.

Microscopic slides of the biopsy specimen showed pleomorphic tumor cells, many of them multinucleated, growing wildly in fibrillar stroma. In some areas formation of osteoid and bone was observed, while elsewhere there was focal deposition of calcium. Mitotic figures and giant nuclei were frequently observed.

Sections from the amputation specimen showed the same type of tumor cells invading the medullary cavity, the cortex, and the muscle adjacent to the fibular head. Cartilagenous stroma was found in some areas, and bone production was noted in many places. The pathological diagnosis was osteogenic sarcoma of the sclerosing type.

Dr. Helwig: Dr. Weaver, would you comment on this case?

Dr. Weaver: This is a reasonably early case of

* Cancer teaching activities aided by a grant from the National Cancer Institute and the Kansas Division of the American Cancer Society.

** Trainee of the National Cancer Institute.

osteogenic sarcoma. The patient had pain and a tumor mass; these, plus the fact that she was in the younger age group and had the x-ray findings that we have seen, caused us to suspect strongly that she had an osteogenic sarcoma. Then the next step in management was to secure a biopsy. My feeling is that a biopsy in no way lessens the patient's chance for life. The only complication to be expected following biopsy of a malignant tumor results from delay in amputation; in that instance, the tumor may burst through the biopsy site. There are some advocates of local resection of malignant bone tumors. I don't belong to that school. I think in rare selected cases it constitutes proper treatment, but in most cases amputation is required. In this case the tumor had extended into the soft tissues. Local resection, though it would have preserved the function of the limb, would also have invited recurrence. Even with amputation, this patient's chances for survival are not good. In over 100 cases that we have followed, there have been only a few survivals. It was once my opinion that cures of osteogenic sarcoma varied inversely with one's diagnostic ability—that is, if a case was "cured," the diagnosis had been wrong. I had not seen an undoubted case of osteogenic sarcoma that survived. I have now seen several. Despite the fact that therapy is unchanged, we have had no deaths in the eight cases of osteogenic sarcomas of the extremities that we have seen since 1945.

Dr. Helwig: Will you present the second case, please, Dr. Litton?

CASE NO. 52-113

Dr. Litton: This 10-year-old white girl was admitted to the hospital complaining of pain in the right shoulder. She had first started to have pain in the shoulder about 3 months prior to admission, but the pain was not severe enough to inhibit her in any of her normal activities. About one week before admission, however, the shoulder became much more painful. She had a poor appetite and had lost a little weight. She had no chills, no fever, and no other joints were involved.

Physical examination showed a girl rather small and thin for her age. There was tenderness over the region of the deltoid insertion of the right humerus and pain with external rotation. There was a firm mass palpable on the anterior surface of the humerus about two inches below the coracoid. There was no redness, and there was no swelling about this area and no regional lymphadenopathy. Laboratory work was essentially negative, and the patient had brought x-rays with her which showed a destructive lesion in the head of the right humerus.

Under general anesthesia a biopsy of the lesion was done, and the area was completely curetted. The postoperative course was uneventful. She was seen again about five months later, and an x-ray of her humerus at this time showed a picture which was rather indicative of malignant tumor. Her subsequent course in the past two months has been entirely uneventful.

Dr. Tice: I have not examined the original films. They were reported as showing an inflammatory process. My comment is that if I had not had that report, I would certainly have considered the postoperative films indicative of tumor, because they show a destructive process without sequestration. The picture is distorted by the surgical procedure. The picture to me is that of a destructive process, no bone production, no sequestration. It looks like a malignant process more than an osteomyelitis.

Dr. Helwig: Dr. Boley, what did the biopsy look like?

Dr. Boley: The microscopic sections showed fragments of necrotic bone and marrow spaces that often contained excessive amounts of fibrous tissue. Scattered lymphocytes, monocytes, plasma cells, eosinophiles, and polymorphonuclear leukocytes were found. Evidence of bone formation was noted in places. Osteoblasts and fibroblasts were uniform in size and shape. Our interpretation was chronic osteomyelitis.

Dr. Helwig: Dr. Weaver, would you tell us about the postoperative course?

Dr. Weaver: Our cultures at the time of operation were negative. There was no subsequent drainage, and the cavity in the humeral head is now almost obliterated; this course confirms the inflammatory nature of the process.

Dr. Helwig: There was never any record, was there, Dr. Weaver, of her having any fever, leukocytosis, or unusual pain suggesting a bout of acute osteomyelitis?

Dr. Weaver: Nothing other than some pain.

Dr. Helwig: Would someone like to comment on the differential diagnosis between Garré's chronic osteomyelitis and some bone tumors, particularly Ewing's, and even osteogenic sarcomas?

Dr. Schloerb: Would one not add syphilis to that list?

Dr. Helwig: Do you mean as a possible diagnosis here? Yes, particularly syphilis can be easily confused with reticulum cell sarcoma. There was an exhibit, I think it was at the meeting of the Academy of Orthopedic Surgery, less than a year ago, wherein that was brought out as a very difficult differential diagnosis, particularly since in syphilis and in some bone tumors they are apt to have the classical osteocopic night pains, which in the old days was thought to be practically diagnostic of syphilis. A great many

of those patients who were questioned had a few mercury treatments and they did pretty well.

Often Garré's sclerosing osteomyelitis is confused with Ewing's endothelioma, because both will have fever and both will have leukocytosis, and pain, and both will have involvement of a fair-sized portion of the shaft at times. In operating upon Ewing's tumor, one is apt to encounter bubbles of purulent appearing material oozing out through the periosteum. Anatole Kolodny[1] emphasized this difficulty in differential diagnosis. The "pus" in Ewing's sarcoma, of course, is sterile; but, with preoperative administration of antibiotics, it isn't uncommon to see "sterile" pus in osteomyelitis. Now we rarely see acute fulminating osteomyelitis with high fever and septicemia.

Dr. Tice, would you like to comment on the differential diagnosis from the x-ray standpoint?

Dr. Tice: I think that Garré's sclerosing osteomyelitis and osteogenic sarcoma are very difficult to differentiate. We have in the past followed a case or two in which there was a sclerosing fusiform type of tumor at the lower end of the tibia which failed to progress like a malignant tumor. It was not biopsied and was thought to be sclerosing osteomyelitis. We have also had one case of so-called "quiet necrosis" or serous osteomyelitis which in some respects resembled this case. There was no clinical picture of osteomyelitis. It had a shaggy type of periosteal bone production at right angles to the shaft. Our first impression was that it was an osteogenic sarcoma. When we got adequate films, we could see the shaggy periostitis involving the entire shaft. It was diagnosed osteomyelitis because of the presence of sequestration. I think Ewing's tumor may be difficult to differentiate from osteomyelitis, clinically and radiologically. In Ewing's tumor our first impression may be osteomyelitis, and later on, when a biopsy is done, it proves to be a tumor.

Dr. Weaver: For a number of years, until the x-ray plates were lost, I had taught my students by showing them a case of a typical "Ewing's tumor" that we had x-rays of. Actually, the case was one of osteomyelitis, but it was the only typical "Ewing's" that I had in the place. It was proved an osteomyelitis at biopsy, and it cleared up. I followed the child for several years.

Dr. Stoddard: I'd like to ask a question about this case. Did this girl have preoperative antibiotic therapy?

Dr. Weaver: She did not.

Dr. Stoddard: I wonder if it is usual or unusual to find this kind of localized, apparently low grade, chronic osteomyelitis in a child, or is that more common in adults; is this an example of what has been called Brodie's abscess, or is that another entity?

Dr. Weaver: It's not exactly uncommon to find these things in a child. I think in this case one might call this a Brodie's abscess.

Dr. Helwig: Let's have the last case, Dr. Litton.

CASE NO. 52-114

Dr. Litton: This case is that of a 21-year-old white man, who was admitted to the hospital complaining of pain and swelling of the lower lateral aspect of the right femur and intermittent aching of the knee joint for six months. This pain became more frequent and more severe until about six weeks before admission, when it became almost continuous. During the month prior to admission, he had noted a loss of about 20 pounds weight, loss of appetite, general malaise, and irritability. Physical examination was negative except for tenderness in the right supracondylar region of the right femur on the lateral aspect. There was moderate swelling of the knee. The laboratory work was essentially negative. Amputation was done in November, 1951, and the postoperative course was uneventful. The patient has remained symptom free.

Figure 1. Case No. 52-114. X-ray of lower end of right femur showing hemispherical tumor mass on lateral aspect with destruction of the cortex and slight new bone formation at the limiting margin. Inset: Photograph (without magnification) of a hematoxylin and eosin stained gross section through the lesion shown by the x-ray.

Dr. Helwig: Dr. Tice, can you tell us about the x-rays?

Dr. Tice: One film (Figure 1) was made at Independence Sanitarium and was interpreted by Dr. Bowser as a malignant bone tumor. Films made in our department occasioned considerable discussion but were reported as showing malignant bone tumor. I couldn't quite see in them the typical picture of osteogenic sarcoma. The cortex is expanded locally, forming a "bubble" on the silhouette of the femur. There is some destruction, I will grant, in the expanded area behind that. I do not see bone production in the soft tissues. The cortex outlines the tumor but appears to contain it. I was inclined to consider it an osteochondroma or possibly an osteochondrosarcoma, but I didn't think it was a classical osteogenic sarcoma. I am afraid I was wrong.

Dr. Helwig: Dr. Boley, what were your findings?

Dr. Boley: The gross specimen (Inset, Figure 1) showed a great deal more than the x-ray film did, in that the cortex was destroyed by an ovoid, soft, friable, dark brown tumor mass measuring 3 by 2 cm. and situated 9 cm. from the proximal line of excision of the femoral shaft.

Microscopic sections (Figure 2) showed spindle-shaped tumor cells associated with multinucleated giant cells and irregular fragments of osteoid and bone bursting through the cortex of the femur and invading the surrounding muscle. Tumor was also found invading the medulla. Again the diagnosis was sclerosing osteogenic sarcoma.

Figure 2. Case No. 52-114. Hematoxylin and eosin stained section of tumor showing disorganized proliferation of osteoblasts with irregular osteoid and new bone formation, scattered giant cells and marked vascularity. X 160.

Dr. Helwig: What was the character of those stromal elements in between the newly-formed bone spicules?

Dr. Boley: Most of these cells were of the spindle cell type; some of them were a little larger, plumper cells.

Dr. Helwig: Were there any embryonic osteoblasts there? How do you differentiate this lesion from fibrous dysplasia?

Dr. Boley: There is too much cellular variation for a simple fibrous dysplasia. We have multinucleated giant cells in some places which probably represent osteoblasts in this tumor.

Dr. Helwig: Well, my reason for bringing it up is that I've seen cases of fibrous dysplasia do exactly the same thing with marked metaplastic bone production. I think the gross picture here is adequate for diagnosis without the sections, and I think it should be re-emphasized that the diagnosis of osteogenic sarcoma may be extraordinarily difficult.

Dr. Weaver, would you tell us about the follow-up on this case?

Dr. Weaver: I saw this chap a month ago. He has his prosthesis, he is back on the job, he is married and has youngsters and has invested in a new Ford car, and so far he's perfectly happy and we are keeping our fingers crossed. We don't know.

Dr. Helwig: Dr. Schloerb, there have been some studies on the etiology of osteogenic sarcoma and the possible role of radon, radium, plutonium and beryllium. Would you like to comment on any of those agents, and any other possible etiologic factors?

Dr. Schloerb: The occurrence of osteogenic sarcoma in individuals with known history of exposure to radioactive materials brings up an interesting question as to whether other people developing this tumor might possibly have been exposed to a radioactive agent at some time in their lives. We have checked several of these tumors for radioactivity, but the results were inconclusive.

Dr. Helwig: At Bethesda at the present time they're gathering data on cases of radium dial painters and others, including a group of people who had drunk large quantities of radioactive water, up around Pennsylvania someplace. There were a number of cases of osteogenic sarcoma in these people who drank this so-called "healing water." You are all familiar, of course, with the dial painters' sarcoma. Are there any other comments that anyone would like to make? Dr. Stoddard?

Dr. Stoddard: Postirradiational osteogenic sarcoma of the vertebrae following the treatment of testicular

tumors which has been reported recently[2, 3] would be in that same category.

Dr. Tice: It's interesting that occasionally patients with Paget's disease will develop osteogenic sarcoma. We had such a case presented at this conference some time ago.[4]

Dr. Helwig: Yes, it has been estimated that 28 per cent of cases of Paget's disease will end up as osteogenic sarcoma.[5] There are two types of osteogenic sarcomas that are usually seen in the older age group: those on the basis of Paget's disease, or chondroma or osteochondroma which becomes subsequently malignant. I have seen two cases of multiple cartilagenous exostoses undergo sarcomatous transformation. And that always brings up the question of prophylactic removal of an osteochondromatous exostosis for fear of its subsequent malignant transformation. How do you feel about that, Dr. Weaver?

Dr. Weaver: Most osteochondromas, particularly if they are unsightly or interfering with function, should be removed. This is especially true if they have a tendency to grow. Surgical removal is not a difficult procedure.

Dr. Helwig: Thank you, Doctor. I might say, in order to re-emphasize the difficulty of diagnosis, that I know of one case that was amputated for scurvy and I know of two cases that were amputated for ossifying myositis, which latter may be extremely difficult to differentiate roentgenologically from osteogenic sarcoma. This is a complex subject, and we have barely vaccinated the superficial surface of it today.

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Film for Rural Areas

A new motion picture film, "A Citizen Participates," has been issued to show how members of a rural community can work together to get a physician. Produced by Centron Corporation and cleared by the A.M.A., the film should be a valuable public relations tool for county medical societies. It may be obtained on loan for showings to various organizations and business groups.

Complete information may be secured from Young America Films, Inc., 18 East 41st Street, New York 17, New York.

BLUE SHIELD

Blue Shield Goals

From time to time an organization should pause and take a look at its goals. Such introspection should be outside the framework of its day-to-day activity, its current problems, and temporary solutions. It should be based on the long view—a reappraisal of early goals, an evaluation of progress, and a recasting of future goals.

What were the early goals of Blue Shield, to what extent have they been achieved, and what should the future targets be?

Before considering these questions it is well to state clearly that the medical profession in Kansas has the full responsibility for the fate of Blue Shield. Blue Shield goals must be set by the doctors, and these goals cannot be realized without the acceptance and co-operation of the vast majority of the profession.

Original Goals. In Kansas, the first goal of those who believed in the Blue Shield idea was to bring the plan into being. Here was a new and still highly experimental idea—actually a controversial idea to many physicians. Looking back, one can now better appreciate the foresight, the imagination, and the vigorous courage it took to tackle a plan as involved as Blue Shield actually was. When we consider how easy it is to wash our hands of a complex problem which involves widespread group action, we must be grateful to the leadership in the Kansas Medical Society, Dr. Barrett A. Nelson and others who devoted so much energy and time to the formation of Kansas Physicians' Service.

A good statement of the problems and goals of Blue Shield is contained in a report Doctor Nelson made in the JOURNAL in May 1944. As chairman of the Medical Economics Committee, he had just returned from a Chicago meeting of the Medical Service Plan Council, a group of physician-sponsored plans then in operation. Doctor Nelson reported: "The conviction was obtained that this rapidly spreading movement truly fills a need which has long existed in the economics of medical practice, making it possible for patients to budget against the financial strain of serious illness.

"The successful plans are remarkably similar in character. It definitely has been found that the public is not ready for a plan for over-all and complete coverage. The costs are necessarily more than the purchaser of a contract is willing to pay, and there is, as yet, insufficient actuarial basis for complete knowledge of proper costs.

"The tentative plan for Kansas would . . . limit

coverage, for the present, to surgery, orthopedics and obstetrics.

"The proposed contract would present a fee schedule furnishing complete payment for the prescribed services for individuals with an annual income below $1,800 (or a family income of $2,400), while those with higher incomes would be expected to pay the difference between the fee schedule and the usual fee for their income levels."

A key to the goal thinking of the planners of Blue Shield lies in the phrase "enabling people to budget the cost of serious illness."

Another notable point is that early goals were properly and necessarily restricted because of lack of actuarial data and financial stability.

Evaluation of Progress. Has Blue Shield achieved its early goals?

Two of the goals were to gain actuarial experience and achieve financial stability. Both have been accomplished.

The original idea of a limited surgical-obstetrical plan has been expanded considerably. Even before Blue Shield started business it added (in hospital) medical benefits. These medical benefits have been improved on two occasions since the first contract.

Since the first contract the following other expansions of coverage have been adopted:

1. X-ray therapy for cancer
2. Isotope therapy for thyrotoxicosis and cancer of the thyroid
3. Removal of $175 maximum for any one illness
4. Removal of $300 maximum for any one year
5. Development of a $5,000 extended benefits rider for polio and eight other diseases. (This is a pioneer step in the direction of full coverage for catastrophic illness.)

Enrollment. In the meantime, Blue Shield has had a gratifying growth in membership, the latest enrollment total being nearly 320,000 members. Kansas Blue Shield today is in a position of strength from which it can now move forward into new concepts of medical economics.

New Goals. What new goals should now be developed for Blue Shield?

Of course, there is the ongoing goal to enroll as many people as possible. But this goal of enrollment can best be achieved through continuous improvement in our program.

Here are some long range possibilities to think about.

1. Extension of coverage for catastrophic illness to include other conditions such as cancer, rheumatic fever, and multiple sclerosis.
2. Higher fee schedules with deductible provisions in order to give maximum protection to the middle income group.
3. Open fee schedule contracts for executive personnel in large employee groups.
4. Lower rate, lower fee schedule contract for low income group which we are not now enrolling.

These broadly defined ideas would need a great amount of study and clarification before they might be adopted. It should be repeated here that the medical profession controls the fate of Blue Shield. Until there is acceptance by doctors, Blue Shield cannot move forward unto new goals.

The thoughts of individual physicians are solicited at this time. You are urged to take time out and write your ideas to the JOURNAL. They will be passed along to the Blue Shield board for consideration.

NEW CHAIRMAN FOR ADVISORY COMMITTEE

Dr. Byron J. Ashley, Topeka, has been named chairman of the Kansas Volunteer Advisory Committee to succeed Dr. Lucien R. Pyle, Topeka, who resigned recently when he became president of the Kansas Medical Society.

Other members of the committee are Dr. Thomas R. Hood and Dr. H. H. Cook, the latter representing dentistry. Assisting in an advisory capacity are Dr. A. R. Bower, for veterinarians, and Miss Irma Law, for nurses.

"This is an advisory committee," Doctor Ashley said, "and as its name implies, it has no official powers of any kind. Our primary duty is to preserve essential civilian health services, so where the induction of a physician would seriously affect a community, we recommend a delay in his orders to provide time for the people to obtain a replacement.

"We also are charged with aiding Selective Service in providing certain health personnel for the armed forces. Therefore, we are required to maintain an up-to-date list of all available doctors in this state.

"Our duties are two-fold. We represent a co-ordinating mechanism between Selective Service and the local community, but our principal concern is to protect the people of this state from serious shortages in services that are provided by physicians, dentists or veterinary physicians."

HEART FELLOWSHIPS AND GRANTS AVAILABLE

American Heart Association fellowships for the 1954-1955 year are now being planned, and applications may be submitted as late as September 15, 1953. Applications for research grants-in-aid must be submitted before December 1, 1953. Information and application blanks may be secured from the association, 44 East 23rd Street, New York 10, New York.

The Diagnosis of Polyarteritis Nodosa: A Review of the Literature and the Presentation of Two Cases*

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Polyarteritis nodosa is a disease of many names possessing more than 15 synonyms.[1] The most frequently used are: periarteritis nodosa, polyarteritis, panarteritis, necrotizing arteritis, primary arteritis, and essential polyarteritis.[2]

Polyarteritis nodosa was first described, microscopically, by Rokitansky in 1853.[3] The first complete description of the disease was made by Kussmaul and Maier in 1866.[4] However, it was Meyer that presented the symptom triad for polyarteritis nodosa (chlorotic marasmus—polyneuritis, polymyositis, and gastrointestinal symptoms.[5] This triad is still descriptive of the most common symptoms encountered today.

ETIOLOGY

The etiology of polyarteritis nodosa has been attributed to many diseases or substances. The most commonly cited "etiological agents" are asthma, rheumatic fever, syphilis, hypertension, tuberculosis, a filterable virus, unknown specific infections, streptococcal infections, stress, and various sensitivity reactions.[3, 6, 7, 8, 9] Currently sensitivity reactions are believed to be the most likely causes.

Some of the substances that have been incriminated are sulfonamides, desoxycorticosterone, anterior pituitary substances, iodine, penicillin, activated ergosterol, histamine, propylthiouracil, Dilantin, and foreign sera.[2, 3, 6, 7, 8, 10, 11, 12]

It is interesting to note that polyarteritis is rarely associated with common forms of allergy in children.[13]

PATHOLOGY

Polyarteritis nodosa is commonly included in the group of so-called diffuse collagen diseases.[16] Some investigators consider it to be pathologically related to Henoch's and Schoenlein's purpuras, rheumatic fever, rheumatoid arthritis, thromboangiitis obliterans, serum sickness, and possibly to Loeffler's and Libmann-Sacks' syndromes.[3, 17] One authority even relates collagen diseases, including polyarteritis, to old age.[18]

Essentially a disease of the medium and small arteries, it affects the media initially and the intima and adventitia later.

Arkin has classified the pathological findings into four stages and has attempted to correlate these with four clinical stages.[15]

1. Beginning. Edema and fibrinous exudate occur within the media along with neutrophil and eosinophil infiltration producing narrowing of the vessel lumen. The clinical finding in this stage, if any, is fever.

2. Acute Inflammatory. In this stage the media, adventitia, and perivascular tissues are invaded by neutrophils, eosinophils, lymphocytes, and plasma cells; there is intimal and subendothelial connective tissue proliferation. Infarcts and thrombi are common. Clinically one encounters fever, chills, leukocytosis, eosinophilia, anemia, and symptoms of organ involvement. In this stage, the most common causes of death are cardiac or renal insufficiency.

3. Granulation. Fibroblastic infiltration of the involved layers and intimal proliferation occur. Aneurysms and vessel rupture are common. The clinical picture presents fever, emaciation, decreasing leukocyte count, anemia, and symptoms of organ involvement. Death from hemorrhage may occur.

4. Healed. The microscopic examination reveals scarred vessels with narrowed lumina and irregular, thickened walls. The entire organ involved may be shrunken and scarred. The patient may display a normal temperature and leukocyte count with tachycardia and symptoms of organ insufficiency.

The incidence of involvement of the various organs is: kidneys, 80 per cent; heart, 70 per cent; liver, 65 per cent; gastrointestinal tract, 50 per cent; mesenteric arteries, 30 per cent; pancreas, 25 per cent; peripheral nervous system, 20 per cent, and central nervous system, 8 per cent.[28] Several other observers present findings similar to these.[23, 35, 39, 51]

INCIDENCE

There have been about 500 cases of polyarteritis reported in the English literature.[19] The ages of the patients ranged from 10 days to 89 years.[19] The highest incidence is between the ages of 20 and 40 years.[16] It was found that only 52 of 484 patients were under 15 years.[19]

Of the above cited 484 cases, the male to female

* This is one of 11 senior theses selected for publication by the Editorial Board from a group of 15 judged the best by the faculty of the University of Kansas School of Medicine.

** Thesis written while the author was a senior student. Dr. Ray is now interning at St. Joseph Hospital, Kansas City, Missouri.

ratio in adults is 2.3 to 1 and in children it is 1 to 1.08.[19]

CASE HISTORIES

Within the past year there were two cases of polyarteritis admitted to the University of Kansas Medical Center. The case histories follow.

Case 1 (ECV). This 47-year-old white male was admitted to the KUMC on January 2, 1952, with a chief complaint of pain, numbness, and swelling of feet and paralysis of right foot.

The patient's symptoms began in November, 1951, with a severe, dull, aching pain along the medial surface of the left leg recurring at about weekly intervals. During December the sole of the left foot became painful and the right lower extremity became affected with a severe burning pain in the popliteal area, swelling, and foot drop. The patient had also noted paresthesias of the left forearm and the palmar surfaces of the first and second fingers.

The patient had had an esophagogastroplasty to correct a congenitally short esophagus and hiatal hernia (1950) and a stomach operation at the age of five.

The important positive findings of the admission physical examination were limited to the lower extremities. There were a moderate amount of calf muscle atrophy bilaterally, swollen feet, and right foot drop. Ankle jerks were absent; there was loss of position sense of the right foot, and decreased pain and temperature sensations of the right sole.

The admission laboratory findings were as follows: urinalysis normal; RBC 4,800,000; Hb. 89 per cent; WBC 17,000 with polys 76 per cent, lymphs 21 per cent, and monos 3 per cent. The routine and special blood chemistry was negative except for a non-protein nitrogen of 42 mg. per cent. The agglutinations, stool examinations, spinal fluid, and liver function tests were negative. The EKG showed a sinus tachycardia; x-rays and EEG were non-contributory.

During the course of hospitalization, the urine remained essentially negative except for varying amounts of proteinuria. The WBC varied from 17,000 with 76 per cent polys to 39,400 with 83 per cent polys. Total circulating eosinophils dropped from 494/cu. mm. to as low as 38/cu. mm., possibly in response to treatment. Renal function tests indicated at least moderate renal insufficiency.

The patient ran a low grade febrile course with moderate tachycardia; blood pressure varied from 150/90 to 200/110. Neurological findings were consistent with a clinical diagnosis of polyarteritis nodosa. Although the first muscle biopsy was negative, the second, a few days later, showed changes characteristic of polyarteritis.

The patient received no benefit from liver extract and thiamin chloride. ACTH produced immediate relief of pain and some decrease of the paresthesias although there was no improvement of the edema and weakness. When ACTH was discontinued for 10 days, the symptoms returned.

The patient was dismissed February 9, 1952, on ACTH, potassium chloride, and Vitamin C.

Case 2 (PR). This 40-year-old white male was first seen for this illness by his local physician on November 14, 1951, complaining of soreness and swelling of the legs.

A painful and tender swelling of the anterior lower right thigh first appeared about the middle of November and lasted several days, leaving slowly. The overlying skin became slightly thickened. About a week later the dorsum and lateral aspect of the right foot became swollen, red and painful. There was no joint involvement although the patient had had migratory joint pain and swelling and severe burning of the feet and fingers since an attack of pericarditis in June, 1951.

Physical examination (November 14) showed the right foot to be swollen, red, edematous (non-pitting), and sensitive to light touch; there was some edema of the right leg and numbness of third and fifth toes. There was slight swelling of the dorsum of the left foot. Knee jerks were sluggish.

The urine showed proteinuria and slight pyuria and hematuria. The sedimentation rate was increased.

The patient returned to his physician after several days in bed. He still had severe leg pain, worse at night. The inflammation of the right leg and foot had subsided although there was tenderness and hypesthesia over the dorsum of the foot. The urine was about the same. A biopsy of the leg muscle was negative.

Examination upon admission to KUMC (December 12, 1951) revealed marked hyperesthesia, in response to light stroking, of the right lower leg and dorsum of the foot and, to a lesser extent, of the left leg. One axillary and a few cervical glands were found to be slightly enlarged.

Extensive laboratory work was done with few abnormalities being found. The urine showed slight proteinuria, pyuria, and cylindruria for four times; blood was positive once. The glucose tolerance exhibited the following: fasting 56 mg. per cent; 1 hour, 195 mg. per cent; 2 hours, 195 mg. per cent; 3 hours, 110 mg. per cent; 4 hours, 68 mg. per cent; and 5 hours, 63 mg. per cent. The first, second, and third hour urine specimens showed positive sugars. The sedimentation rates were slightly to moderately elevated. Routine and special blood chemistry tests, renal function tests, spinal fluid examinations, skin tests, hepatic function tests, and other examinations were all within normal limits. The chest x-ray showed emphysema and increased hilar shadows of an infiltrative nature; other x-rays were non-contributory.

Biopsies of pectoral muscle and axillary lymph node were negative.

The patient's short hospital course was uneventful; the blood pressure never exceeded 160/90 (admission), and the temperature was always under 99° F.

The patient was dismissed (December 19, 1951) with the most likely diagnosis being polyarteritis nodosa. His local physician was advised to use ACTH or cortisone.

CLINICAL SIGNS AND SYMPTOMS

The signs, symptoms, laboratory, and x-ray findings most frequently encountered in the cases reported by six investigators are listed in Chart I.

To list completely all of the signs and symptoms found in patients having polyarteritis would be futile. Almost every sign or symptom has been found at some time in patients afflicted, yet few are encountered consistently enough in all cases to be considered of diagnostic value.

The pathology of polyarteritis shows that it is a disease of the vascular system resulting in occlusion and/or hemorrhage, the occlusive phenomena being most prevalent. Therefore, the findings are those produced by ischemia of one or more organs or systems.

The onset may be rapid or insidious, progressing over days or months. About 18 per cent to 25 per cent of the patients have a history of allergic manifestations.[24]

The presenting symptom will most likely be that of pain, particularly abdominal pain or discomfort.[25] In one series of 30 patients, gastrointestinal symptoms occurred in 75 per cent.[26] About half of the patients complaining of pain had it in the epigastrium or right upper quadrant.[26, 27] Many laparotomies have been performed because of abdominal discomfort and vomiting frequently accompanying it.[28]

The involvement of any of the intraabdominal organs as well as the supporting structures and the abdominal wall has resulted in findings resembling peptic ulcer, appendicitis, gastrointestinal perforation, peritonitis, pancreatitis, ruptured spleen, and others. If the abdominal discomfort is of ischemic origin, morphine will not provide adequate relief.[27] This may serve to assist in differentiating polyarteritis from most inflammatory processes.

Other abdominal and gastrointestinal signs and symptoms that may be present are nausea, vomiting, constipation, diarrhea, distention, melena, jaundice, splenomegaly, and hepatomegaly, the latter usually resulting from congestive heart failure.[3, 7, 19, 27] A detailed list of abdominal symptoms does not advance the diagnosis greatly.

Renal findings are commonly a part of the picture; renal and genitourinary involvement occurs in practically every case.[21] The association of renal symptoms along with polyneuritis is suggestive of polyarteritis.[29]

CHART I

SIGNS, SYMPTOMS, AND LABORATORY FINDINGS ENCOUNTERED IN 10 PER CENT OR MORE OF PATIENTS HAVING POLYARTERITIS NODOSA

Source (Ref.)	4	20	21	22	14	23	
Total Patients	7	30	6	17	20	177	257
Sign, Symptom							*Total*
Fever	7	21	6	12	9	143	198
Gastrointestinal		23(a)					23
pain[1]			5	11	3	99	118
nausea-vomiting			4	6		57	67
jaund.-hep'meg.			3	11		21	35
Renal							
albuminuria	5		4	14	6	115	144
hematuria	4	17	4		1	85	111
azotemia	4	15			2	23	44
Nervous System		14(b)			2(c)	46(d)	62
coma			3			37	40
sensorial[2]					16	60	76
eye signs			1(e)	1(f)	1(g)	41	44
headache			3		4	46	53
neuritis, periph.	5				17	87	109
Muscular							
atrophy					10	35	45
weakness	5		6		17(h)	80	108
Cardiovascular		17(i)			8(j)		25
HBP		20(k)	3	14	11	94	142
dyspnea			4	8		71	83
cyanosis						35	35
tachycardia			5	16	13	85	129
edema[3]	4			11		87	102
Pulmonary							
cough				3		64	67
pain						30	30
Skin							
nodules		4	3	2	2	41	52
other lesions[4]		6		2		34	42
Joints							
arthritis						60	60
Blood Findings							
anemia		19	6		9	85	119
leukocytosis		24	6	13	14	129	186
eosinophilia	4	5		1	5	57	72
sed. rate		13		17			30
Weight Loss	4	5		9	14	78	110
Allergic History						37	37
Lymphadenopathy			2	2		19	23

Key to Notes in CHART I:

1. Including abdominal pain.
2. Paresthesias, anesthesias, etc. (?)
3. Edema, regardless to etiology (cardiac, renal, inflammatory, etc.).
4. Purpuras, petechiae, rash, etc.
a. No particular gastrointestinal symptom specified.
b. Hallucinations, disorientation, coma, etc.
c. Focal brain signs.
d. Central nervous system involvement.
e. Blindness.
f. Hemianopsia.
g. Papilledema.
h. Generalized and local.
i. "Cardiac symptoms."
j. Hypertrophy, arrhythmias, murmurs.
k. Blood pressure exceeds 150/90.

Usually edema and slight or profuse hematuria direct attention to the renal system.[29, 30]

Nocturia, cylindruria, high blood pressure, and edema may be present, simulating acute nephritis.[21] Renal colic may also be present.

Microscopic urinalysis often shows the unusual combination of erythrocytes, erythrocyte, fatty and blood casts.[32] Functional tests often show evidence of increasing damage.[29]

When the heart is involved, precordial pain, angina, palpitation, dyspnea, and cough are frequently present.[7] However, Boyd believes that angina is uncommon compared to the frequency of coronary artery involvement (60 per cent).[29] Tachycardia, hypertrophy, systolic murmurs, and peripheral edema are commonly encountered.[7] Tachycardia, present in 65

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Comprising the reports published in the literature to date which give specific facts and figures of the results of treatment

AUTHORS	No. of Patients	Chronic, Resistant to Other Therapy	TYPES OF ULCERS				RELIEF OF SYMPTOMS (Chiefly Pain)				Surgery or Complications[1]	Side Effects Requiring Discontinuance of Drug[2]	EVIDENCE OF HEALING			
			Duodenal	Jejunal	Stomal	Gastric	Good	Fair	Poor	No Report			Complete	Moderate	None	No Report
Grimson, Lyons, Reeves	100	100	93	7			80	11	4		5		47		19	29
Friedman	15	15	14			1	5		4	6[3]			2			13
Bochgaard, Nielsen, Bang, Gruelund, Tobiassen	26	26	21			5	16	4	6				8	6	12	
McHardy, Browne, Edwards, Marek, Ward	162		162				136	12	11		3	1	14	9	7	129
Segal, Friedman, Watson	34	34	34[4]				14	13			7	2	5		8	14
Brown, Collins	117	99	117				97	7	8		5	8	55	9	8	40
Asher	77		65		7	5	52	9	16			16		9	21	47
Rodriguez de la Vega, Reyes Diaz	5	4	5				4		1					3	2	
Winkelstein	116	116	102	8		6	102		14				53		18	45
Hall, Hornisher, Weeks	18	18	18				11		1	6[5]			18			
Maier, Meili	38	38	24			14[6]	27	7	4[7]				10	2	5	21
Meyer, Jarman	25	18	25				21		4							25
Polk, Fromm	37	37	37				33	3	1				33	3	1	
Plummer, Burke, Williams	41	41	41				36		5				38		3	
McDonough, O'Neil	104	100	104				63	10	31			11	4		11	89
Brodors	60	60	58		1	1	35	19	6				10	1	49[8]	
Legerton, Texter, Ruffin	11		11				11									11
Holoubek, Holoubek, Langford	76	69	76				35	27	10		4	10	26		10	36
Ogborn	42		39	2		1	42[9]									42
Shaiken	48	48	48				33	10	3		2		33	10	3	
Johnston	145	145	145				143		2			2	143		2	
Russell, Knox, Stephenson	146		141			5	146					4[10]	53			93
TOTALS	1443	968	1380	17	8	38	1142	132	131	12	26	54	552	52	179	634
PERCENTAGES		67.8	95.6	1.2	0.6	2.6	81.3	9.4	9.3			3.7	70.5	6.6	22.9	

1. Not included in tabulations.
2. Included in "Relief of Symptoms" as "Poor" and in "Evidence of Healing" as "None."
3. Four had no symptoms when Banthine therapy was begun.
4. Of which seven were penetrative lesions and five partially obstructive.
5. No symptoms were present in four.
6. Two with symptoms only; no demonstrable ulcer.
7. Three were psychopathic patients and one had a ventricular ulcer of the lesser curvature.
8. Roentgen findings after treatment period of two weeks; forty-seven had duodenal deformity.
9. All returned to work within a week.
10. In these four, after relief of symptoms, Banthine was discontinued because of urinary retention.

During the past three years, more than 250 references to Banthine therapy in peptic ulcer and other parasympathotonic conditions have appeared in medical literature. Of these reports, 22 have presented specific facts and figures on the results of treatment in a total of 1,443 peptic ulcer patients, 67.8 per cent of whom were reported as chronic or resistant to other therapy. These results are tabulated above and show:

"Good" relief of symptoms was obtained in 81.3 per cent of the 1,405 patients on whom reports were available.

"Complete" evidence of healing was obtained in 70.5 per cent of the 783 patients on whom reports were available.

In all but 9.3 per cent, relief of pain was "good" or "fair." In all but 22.9 per cent, evidence of healing was "complete" or "moderate."

During treatment, 26 patients required surgery or developed complications other than ulcer which required discontinuance of the drug before results could be evaluated.

Of the remaining 1,417 patients, only 3.7 per cent experienced side effects sufficiently annoying to require discontinuance of the drug.

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per cent of the patients, is out of proportion to the temperature and has been considered a diagnostic point.[29]

Hypertension is a common occurrence; its sudden appearance is particularly suggestive of polyarteritis.[29] There may be some etiological relationship between hypertension and polyarteritis, the hypertension being the cause of the polyarteritis.[33]

Some authors call attention to the fact that hydropericardium, alone or as a part of a polyserositis, is common.[29]

One of the most common manifestations of polyarteritis is the presence of peripheral nerve involvement. The most frequent symptoms are pain, weakness, nerve trunk tenderness, paresthesias, anesthesias, and decreased or absent tendon reflexes.[34] The peripheral neuritis is usually in the lower extremities but can be anywhere and may have root distribution.[8] It is usually remittent and asymmetrical.[8]

The motor nerves as well as sensory nerves can be involved, and this may be the reason that muscular weakness is out of proportion to neurological signs.[34]

Muscles are not affected unless the arteries show local changes.[34] This apparently accounts for frequent atrophy and tenderness, aggravated by use and improved with rest.[35] It is interesting to note that a patient who had had a splanchnectomy for hypertension experienced no more pain except when his blood pressure rose to near its formerly hypertensive levels.[36]

The presence or absence of neuritis appears to be unrelated to age, sex, or clinical course of the disease.[37]

Central nervous system symptoms occur frequently. The most common manifestations are convulsions, generalized or Jacksonian, appearing in no less than 15 per cent.[39] Convulsions appear more frequently in younger groups.[38] Involvement of cranial nerves has been recorded several times.[8]

Joint symptoms are not at all rare.[34] Fisher and Gilmore affirm that joint manifestations ranging from arthralgias to migratory arthritis are frequent.[3] However, Lowman states that synovitis and arthritis are uncommon because of collateral circulation.[25] In any event, the joint pain usually fades into the background as the syndrome progresses.[34]

Skin lesions, of one form or another, are frequently encountered in polyarteritis; they are important because of biopsy possibilities.[40] Besides the lesions there is also an extremely common pallor associated with emaciation, the chlorotic marasmus of Myer.[40]

Apparently there are two forms of skin lesions: the cutaneous form which is rare, chronic, and has a favorable outcome, and the subcutaneous form (periarteritis subcutis) which is more common and equally chronic and favorable.[1]

The majority of the skin (cutaneous) lesions are of the hypersensitivity type (i.e., urticarial, purpural, or erythematous) although papular rashes, vesiculation, and bullous eruptions may be seen.[8] The pure form of cutaneous polyarteritis is characterized by "livido racemosa" and formation of dermal or subcutaneous nodules.[41]

The description of livido racemosa supplied by Wertheim is: ". . . livid, arborescent branched skin markings which fade into the surrounding skin without sharp demarcation. Usually thickened, elevated blue-red main trunks and less intensely colored lateral branches of first, second and third order fading into normal skin is evident."[41] This must be differentiated from livido reticularis and livido reticularis peristans.[41] (According to Dorland, livido racemosa and livido reticularis are synonymous.[42])

Skin lesions may be either single or multiple and are usually painful to pressure.[43] The overlying skin may be normal, appear translucent, edematous, or display red or violet colors.[40] They may appear in crops, usually persisting 48 to 72 hours, accompanied by profound weakness or temperature rise.[40, 44] Nodules may appear along superficial arteries and may pulsate.[9] In a survey of 44 pediatric patients, nodules appeared in only 4.5 per cent, other lesions in 61 per cent.[45]

The order of involvement of the skin is: forearm, chest, legs, abdomen, thighs, face, ankles, back, digits, soles, scalp, scrotum, and tongue.[40]

The lungs appear to enjoy a remarkable exemption from polyarteritis.[1] The most frequently encountered symptom is that of chronic cough.[46] Pain may be present if the pleura is involved, and hemoptysis may occur if tracheal ulceration or arterial rupture occurs.[47]

The x-ray picture is not typical. It may be one of pneumonitis, miliary tuberculosis, infarction, emphysema, asthma, bronchitis, or bronchopneumonia.[7, 9, 47] Infiltration from the hilar areas is as "typical" as any.[7] The diagnosis is suggested if one sees, on serial x-rays, the regression of earlier lesions and the progression of others, with or without the development of new ones.[47]

Laboratory findings in polyarteritis are limited almost entirely to the blood count. As a rule hypochromic anemia, mild compared to the pallor of the skin, is present.[1] It is unusual to find an erythrocyte count of less than 2,000,000 or a hemoglobin of less than 40 per cent. Leukocytosis is the rule with counts ranging from 10,000 to 60,000 with the average being 25,000 and, usually, a shift to the left.[21] Eosinophilia, cited as being characteristic, is not often present. Of 300 cases reviewed, 18 per cent had eosinophils; of those, 94 per cent had bronchial asthma.[24] The sedimentation rate is usually decidedly increased.[1]

Lepow et al.[48] noticed a precipitation phenomenon

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in six of their patients, three of whom had polyarteritis proved by biopsy or autopsy and one each of clinically diagnosed cirrhosis, subacute bacterial endocarditis, and septicemia. They noticed the formation of a precipitate in the blood serum after it had stood 24 hours at 4C; this was not due to "cold fractions" or "cryoglobulins." The precipitate formed when there was a reversal in the albumen:globulin ratio, an azotemia, and when the cephalin flocculation test was strongly positive. It is interesting to note that Erich reports that there is a rise of gamma globulin in polyarteritis.[49]

TREATMENT

Treatment will not be discussed here other than to say that it has been notably unsuccessful and that cortisone and/or ACTH holds the best promise at present.

PROGNOSIS

The prognosis of polyarteritis is poor. However, some authorities believe that it is not fatal as often as previously reported.[19] For a disease as widespread and as variable as this one, it is quite conceivable that misdiagnosed or subclinical cases could often occur.

Perhaps the best prognosis of the disease could be determined from the figures compiled by Boyd in a review of 150 cases.[5] Of these, he found the duration of illness to range from 1 week (4 patients) to over 4 years (3 patients) with the majority (90 patients) in the one- to four-month range; seven apparently recovered.

The causes of death in 128 patients were: nephritis, 28; cardiac failure, 31; perirenal hemorrhage, 15; bronchopneumonia, 11; peritonitis, 9; hepatic hemorrhage, 6; cerebral hemorrhage, 5; and 10 other causes, 16.[1]

DISCUSSION

Polyarteritis nodosa is a relatively rare disease; however, it appears that there may be many subclinical or misdiagnosed cases that obviously have not been reported.

Positive antemortem diagnosis can be made only by muscle biopsy. Even by biopsy of a large piece of muscle from the tender areas, only about 25 per cent of the tests are positive.[14] It appears that pectoral muscles produce the highest percentage of positives.[4]

The onset of the disease is varied; the provisional diagnoses in 50 unselected cases ranged from generalized infection (27 patients) to Wilson's disease (1 patient).[50] The usual onset resembles a nonlocalized infectious disease.[1]

There is a certain fundamental picture that should cause suspicion of polyarteritis. This is a picture of fever, malaise, asthenia, and weight loss in the absence of known infections, parasitic infections, neoplastic, metabolic, or congenital diseases. Multiple diagnoses are common.

There are signs and symptoms that often appear at some time in the course of the disease that should serve to make one suspicious of polyarteritis. Often there are signs or history of recent allergic reactions; this may become of major importance if some of the following manifestations are encountered:

1. Skin manifestations, particularly of the sensitivity type if accompanying systemic disease.
2. Asymmetrical neuritis, with paresthesias and anesthesias; its importance increases if there is also asymmetrical myalgia, paralysis, or muscle weakness.
3. Abdominal pain, particularly if accompanied by gastrointestinal disturbances and not characteristic of more common intraabdominal pathology.
4. Renal signs characteristic of glomerulonephritis; albuminuria and hematuria are often present.
5. Hypertension is a frequent finding, becoming more important if it appears suddenly.
6. Tachycardia out of proportion to temperature, the latter almost always elevated to a slight degree.
7. Usually a hypochromic anemia and a neutrophilic leukocytosis is present; eosinophilia is present if asthma is also present.

SUMMARY

1. Two new cases of polyarteritis are presented; one was confirmed by biopsy and the other presented the "typical" clinical picture.
2. A brief review of recent literature is reported with emphasis upon the antemortem diagnosis of polyarteritis nodosa.
3. Some of the clinical signs and symptoms most commonly encountered in polyarteritis are stressed.
4. The only certain means of diagnosing polyarteritis, antemortem, is by muscle biopsy; accuracy is approximately 25 per cent.

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BOOK REVIEWS

Surgery of the Pancreas. By Richard B. Cattell and Kenneth W. Warren. Published by W. B. Saunders Company, Philadelphia. 374 pages, 100 figures. Price $10.

The authors state that this book is based on experience with more than 1,000 patients with surgical diseases of the pancreas treated at the Lahey Clinic. In addition to a study of the material in the clinic, an adequate review of the literature has been made and the references are appended at the end of each chapter.

The first chapter is devoted to the anatomy and physiology of the pancreas. Succeeding chapters discuss congenital malformations of the pancreas, acute pancreatitis, chronic relapsing pancreatitis, pancreatic cysts, pancreatic injuries, islet cell adenomas and hyperinsulinism, carcinoma of the pancreas and periampullary area, and total pancreatectomy.

In the chapter on acute pancreatitis there is a short historical note followed by a thorough review of the etiology, pathology, symptomatology, methods of examination, and medical and surgical treatment. That difficult subject, chronic relapsing pancreatitis, is very well presented. The modern treatment of pancreatic cysts, the medical and surgical aspects of hyperinsulinism, and the management of pancreatic injuries, are all ably presented.

The critical study of carcinoma of the pancreas and periampullary area, with the authors' results of treatment, is to be praised. Five of 32 patients (16 per cent) who had pancreatoduodenectomies for carcinoma of the head of the pancreas, ampulla of Vater, duodenum and distal end of the common duct, have survived from 5 to 9 years.

The text is adequately illustrated with good drawings, roentgenograms, and charts.

This book is not a monograph for surgeons only but would be of great value to any physician who is called upon to diagnose and treat diseases of the pancreas.—*T. G. O.*

An Atlas of Surgical Exposures of the Extremities. By Sam W. Banks and Harold Laufman. Published by W. B. Saunders Company, Philadelphia. 391 pages, 552 illustrations in 179 plates. Price $15.

This is a comprehensive atlas of surgical incisions of the extremities giving in detail the surgical exposures and major structures encountered in any major operation. This volume will be extremely valuable to the surgeon who operates on the extremities

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since some of the simplest, as well as the more complicated incisions, have been included. Any surgeon who has a great deal of traumatic or elective extremity surgery will find this a refreshing and interesting pictorial representation of regional anatomy.—*C. H.*

Living with Cancer. By Edna Kaehele. Published by Doubleday and Company, Inc., Garden City, New York. 160 pages. Price $2.00.

The author, alive six years after being told by two physicians that she had a malignancy, tells the story of her thoughts and experiences during that time. Although the site of the neoplasm is not divulged, the reader can presume only that its origin may be in the uterine cervix, because of the treatment received. At the same time there is no indication that biopsy confirmation has been carried out in this patient; however, the course of the patient makes it entirely possible that a malignancy does exist in fact.

This story in essence illustrates the devastating effect of "fear" on physical and mental stability, and how the overcoming of that fear, through faith in herself as well as her religion, can renourish a starving body back to reasonably good health. The book is fairly well written and is short enough that the repeated effects of introspection and pity soon turn to an enthusiastic attempt at guidance of others. —*S. R. F.*

Essentials of Infant Feeding for Physicians. By Herman Frederic Meyer. Published by Charles C Thomas, Springfield, Illinois. 252 pages, 10 illustrations. Price $7.75.

The author amply illustrates the seeming complexity of feeding normal babies by using as a frontispiece a photograph of 107 different commercial preparations available for making infants' formulas. He further emphasizes the physician's need for a concise and practically applicable source of information on the principles of infant feeding by stating that, "Sixty to 80 per cent of the average pediatric practice consists of the routine feeding of normal babies." Whether the book adequately fulfills the physician's need in this field is open to question.

The index is excellent, befitting a book designed for rapid reference. The bibliography is extensive. Unfortunately, references are listed alphabetically instead of by subject, which would seem the more useful arrangement in a reference book. The section on description and evaluation of proprietary formula preparations is remarkably complete and constitutes useful reference material. Equally useful material, a classification and evaluation of proprietary vitamin preparations, is completely neglected.

Some of the text, such as the pages berating vitamin advertising, does not seem necessary in a book designed for "brevity and compactness." At the end of the text there is a 37-page appendix consisting of weight and measure equivalents, nutritional requirements and their food sources, and other material, some of it not directly pertinent to infant feeding (e.g. parenteral fluid administration, newborn nursery regulations). The book is written in a controversial style which at times does not clearly convey the author's meaning. Some controversial problems, such as the feeding of infants with diarrhea, are treated with perhaps an unjustified dogmatism. The author is best when drawing upon his own pediatric experience to advise about techniques of infant feeding and parent education. Feeding fads are sensibly discussed.

Though containing much useful reference material, the book is probably better suited as a resumé and overall introduction to the problems of infant feeding than as the handy pocket reference that was intended.—*C. A. M.*

ANNOUNCEMENTS

The 39th annual clinical congress of the American College of Surgeons will be held October 5 through 9, 1953, at Chicago, with headquarters in the Conrad Hilton Hotel. Surgical techniques and new developments in surgery will be covered, and the program will include formal scientific papers, panel discussions, symposia, postgraduate courses, surgical forums, medical motion pictures, ciné clinics, color television, and exhibits.

The American Congress of Physical Medicine and Rehabilitation will hold its 31st annual scientific and clinical session at the Palmer House, Chicago, August 31 through September 4. Sessions are open to all members of the American Medical Association. Full information may be obtained from the Congress, 30 North Michigan Avenue, Chicago 2, Illinois.

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in cooperation with the respective state chapters and the staffs and faculties of the local hospitals and medical schools, will sponsor the 8th annual postgraduate course on diseases of the chest at the Hotel Knickerbocker, Chicago, September 28 through October 2.

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Surgical Technic, Surgical Anatomy & Clinical Surgery, Four Weeks, starting October 26
Surgical Anatomy & Clinical Surgery, Two Weeks, starting August 17, November 9
Gallbladder Surgery, Ten Hours, starting October 26
General Surgery, One Week, starting October 5
Surgery of Colon & Rectum, One Week, starting September 21
Basic Principles in General Surgery, Two Weeks, starting September 21
Thoracic Surgery, One Week, starting October 12
Esophageal Surgery, One Week, starting October 19
Breast & Thyroid Surgery, One Week, starting October 26
Fractures & Traumatic Surgery, Two Weeks, starting October 26
GYNECOLOGY—Intensive Course, Two Weeks, starting September 21
Vaginal Approach to Pelvic Surgery, One Week, starting August 31
OBSTETRICS—Intensive Course, Two Weeks, starting October 5
DERMATOLOGY—Intensive Course, Two Weeks, starting October 19
MEDICINE—Electrocardiography & Heart Disease, Two Weeks, starting October 12
Intensive General Course, Two Weeks, starting September 28
Gastroenterology, Two Weeks, starting October 26
Allergy, One Month and Six Months, by appointment
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A similar postgraduate course will be held at the Hotel New Yorker, New York City, November 2-6. Tuition for each is $75. Details may be secured from the American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

The American Urological Association again this year is offering an award of $1,000 for essays on the result of clinical or laboratory research in urology. Competition is limited to urologists who have been in such specific practice for not more than 10 years and to men in training to become urologists. Particulars of the contest may be secured from William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. The contest closes February 1, 1954.

A sectional meeting of the American College of Surgeons will be held in London, England, May 17-19, 1954, at the invitation of the surgeons of England who are honorary fellows. All physicians are invited to attend, and to be present also at a meeting of the Association of Surgeons of Great Britain and Ireland, to be held in Leeds, May 13 to 15, and a session of the International Society of Surgery, in Paris, immediately after the London meeting.

The second International Congress of Cardiology will be held in Washington, D. C., September 12-15, 1954. It will be immediately followed by the annual scientific sessions of the American Heart Association, September 16-18.

Physicians in Honolulu, Hawaii, will be hosts to the Sixth Pan-Pacific Surgical Congress, October 7-18, 1954, in Honolulu. The association office has been appointed travel agent for those wishing to attend. Information may secured from F. J. Pinkerton, M.D., Pan-Pacific Surgical Association, Suite 7, Young Hotel Building, Honolulu, Hawaii.

The National Gastroenterological Association will hold its annual convention and scientific session at the Biltmore Hotel, Los Angeles, October 12-14. The program will include a symposium on cirrhosis of the liver, peptic ulcer, diseases of the large bowel, and latest developments in cancer research. The sessions are open to all physicians without charge.

Immediately afterward, October 15-17, the association's annual course in postgraduate gastroenterology will be given at the Biltmore Hotel and the College of Medical Evangelists in Los Angeles. The course will be directed by Dr. Owen H. Wangensteen of Minneapolis and Dr. I. Snapper of Chicago, assisted by a faculty from the medical schools of the Los Angeles vicinity. Only those who have matriculated in advance may attend. Details may be secured from the association, 1819 Broadway, New York 23, New York.

The Arthritis and Rheumatism Foundation is offering research fellowships in the basic sciences related to arthritis as follows: (1) Predoctoral fellowships ranging from $1,500 to $3,000, tenable for one year with prospect of renewal; (2) Postdoctural fellowships ranging from $3,000 to $6,000, tenable for one year with prospect of renewal; (3) Senior fellowships for experienced investigation, with an award of $6,000 to $7,500 per annum, tenable for five years. The deadline for applications is November 1, 1953. Information may be secured from the foundation, 23 West 45th Street, New York 36, New York.

The South Dakota Board of Medical Examiners has announced the passage of legislation creating an annual registration fee for licensees in that state in the amount of $2.00, effective January 1, 1954. Those licensed in that state and wishing to retain such licensure are asked to write the board, 300 First National Bank Building, Sioux Falls, South Dakota.

The American Goiter Association again this year offers a prize award of $300 and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The essays, not exceeding 3,000 words in length, may cover either clinical or research investigations. Complete information may be secured from Dr. John C. McClintock, 149½ Washington Avenue, Albany 10, New York.

Public Relations Institute

Courses in medical public relations will be offered at the A.M.A.'s second Public Relations Institute to be held September 2 and 3 at the Drake Hotel, Chicago.

Designed primarily for lay public relations employees of state and county medical societies, the program will include forums presented by societies in co-operation with local newspapers, tips for public relations programs on limited budgets, and the use of television in public relations. Films available for use by local medical societies will be previewed.

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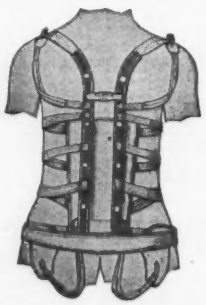

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